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THE BOSTON

Medical and Surgical

JOURNAL.

TRANSACTIONS

OF THE

AMERICAN PEDIATRIC SOCIETY

FOURTEENTH SESSION

HELD AT BOSTON, MAY 26, 27, AND 28, 1902.

WITH ADDENDA

FROM THE THIRTEENTH SESSION

AND AN

INDEX, VOLS. I.–XIV., INCLUSIVE

EDITED BY

WALTER LESTER CARR, M.D.

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CONTENTS.

List of Presidents v

List of Officers for the Year 1902 v

List of Meeting Places v

List of Officers for the Year 1903 vi

List of Members vi

List of Deceased Members viii

Minutes of the Fourteenth Annual Meeting 1

Presentation of a Loving Cup to Dr. Rotch 6

I. Presidential Address. Development the Keynote of Pediatrics. By W. S. CHRISTOPHER, M.D. 9

II. Intussusception, Clinical Remarks. By FRANCIS HUBER, M.D., with Surgical Comments by JOHN F. ERDMANN, M.D. 17

III. Local Variation in the Mortality from Summer Diarrhea. By HENRY DWIGHT CHAPIN, M.D. 34

IV. A Case of Chondrodystrophy Fetalis. By JOHN LOVETT MORSE, M.D. 42

V. The Management of Rheumatic Children. By FLOYD M. CRANDALL, M.D. 59

VI. The Use of the Term "Enanthem." By F. FORCHHEIMER, M.D. 73

VII. Report of a Case of Extreme Enlargement of the Spleen with Anemia. By SAMUEL McC. HAMILL, M.D. 77

VIII. Some Remote Diseases Arising from Tonsilar Infection. By F. FORCHHEIMER, M.D. 91

IX. Conclusions as to the Treatment of Tuberculous Peritonitis in Children. By THOMAS MORGAN ROTCH, M.D. 102

X. Two Cases of Umbilical Fistulæ, Due to Tuberculous Peritonitis. By GEORGE N. ACKER, M.D 110

XI. Typhoidal Appendicitis in Children. By A. SEIBERT, M.D. 114

XII. Chronic Parenchymatous Nephritis in a Child Treated by Renal Decapsulation (Edebohls' Operation). By AUGUSTUS CAILLÉ, M.D. 120

XIII. Diphtheria, With and Without Antitoxin; 159 Cases. By CHARLES GILMORE KERLEY, M.D..... 124

XIV. Some Remarks on Intubation in Diphtheria. By JOHN H. MCCOLLOM, M.D..... 130

XV. The Management of the Fat Percentage in Feeding Difficult Cases. By THOMPSON S. WESTCOTT, M.D..... 137

XVI. The Proteids of Milk. By HENRY DWIGHT CHAPIN, M.D..... 149

XVII. Septic Endocarditis. By SAMUEL S. ADAMS, M.D.. 160

XVIII. The Effects of Tight Diapers. By A. C. COTTON, M.D..... 174

XIX. Pilocarpin in the Treatment of Scarlet Fever. By E. W. SAUNDERS, M.D..... 181

XX. A Case of Autointoxication with Neutral Magnesium Phosphate in the Urine. By CHARLES GODWIN JENNINGS, M.D..... 184

XXI. Clinical Observations on Circulatory Failure in Acute Infectious Disease. By AUGUSTUS CAILLÉ, M.D..... 190

XXII. Sudden Death in an Infant Six Months Old Due to Compression of Large Blood Vessels by an Enlarged Thymus Gland. By AUGUSTUS CAILLÉ, M.D..... 194

XXIII. Report of a Case of Sinus Thrombosis Resulting in Extensive Cerebral Hemorrhage in an Infant Fifteen Days Old. Sigmoid Fusion of the Kidneys. By SAMUEL MCC. HAMILL, M.D..... 196

XXIV. Note on the Temperature Curve of Acute Croupous Pneumonia in Children. By CHARLES GODWIN JENNINGS, M.D..... ... 201

XXV. A Case of Pneumonia, Probably Due to Streptococcus Infection. By WALTER LESTER CARR, M.D..... 205

XXVI. Diphtheria with Persistent Trismus and Opisthotonos, Escherich's Pseudotetanus. By IRVING M. SNOW, M.D..... 210

1901 PAPERS.

I. Congenital Absence of the Abdominal Muscles, With Distended and Hypertrophied Urinary Bladder. By WILLIAM OSLER, M.D. 223

II. The Place of Cereals in Infant Feeding. By HENRY DWIGHT CHAPIN, M.D..... ... 229

III. A Contribution to the Pathological Anatomy of Sporadic Cretinism. By FREDERICK A. PACKARD, M.D., and ALFRED HAND, Jr., M.D..... 245

PRESIDENTS.

1889. A. Jacobi, M.D.
1890. J. Lewis Smith, M.D.
1891. T. M. Rotch, M.D.
1892. Wm. Osler, M.D.
1893. A D. Blackader, M.D.
1894. John M. Keating, M.D.
1895. F. Forchheimer, M.D.
1896. Joseph O'Dwyer, M.D.
1897. Samuel S. Adams, M.D.
1898. L. Emmett Holt, M.D.
1899. Wm. P. Northrup, M.D.
1900. Henry Koplik, M.D.
1901. Wm. D. Booker, M.D.
1902. Wm. S. Christopher, M.D.
1903. J. P. Crozer Griffith, M.D.

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C. P. Putnam, M.D.,
F. Forchheimer, M.D.,
J. C Wilson, M.D.,
F. M. Crandall, M.D.,
T. M. Rotch, M.D.

MEETING PLACES.

1888. Washington, D. C. (Organization), September 18.
1889. Washington and Baltimore, September 20 and 21.
1890. New York, June 3 and 4.
1891. Washington, September 22–25.
1892. Boston, May 2, 3, and 4.
1893. West Point, N. Y., May 24, 25, and 26.
1894. Washington, May 29 and June 1.
1895. Virginia Hot Springs, May 27, 28, and 29
1896. Montreal, May 25, 26, and 27.
1897. Washington, May 4, 5, and 6.
1898. Cincinnati, June 1, 2, and 3.
1899. Deer Park, June 27, 28, and 29.
1900. Washington, May 1, 2, and 3.
1901. Niagara Falls, May 27, 28, and 29.
1902. Boston, May 26, 27, and 28.
1903. Washington, May 12, 13, and 14.

OFFICERS, 1903.

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Secretary.....Samuel S. Adams, M.D.
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F. Forchheimer, M.D., T. M. Rotch, M.D.,
J. C. Wilson, M.D., F. A. Packard, M.D.

MEMBERS.

Acker, George N., M.D.....913 Sixteenth Street, Washington
Adams, Samuel S., M.D.....1 Dupont Circle, Washington
Baines, Allen, M.D.....194 Simcoe Street, Toronto
Blackader, A. D., M.D.....236 Mountain Street, Montreal
Booker, William D., M.D.....208 West Monument Street, Baltimore
Buckingham, E. M., M.D.....346 Marlborough Street, Boston
Caillé, Augustus, M.D.....753 Madison Avenue, New York
Carr, Walter Lester, M.D.....68 West Fifty-first Street, New York
Chapin, Henry Dwight, M.D.....51 West Fifty-first Street, New York
Christopher, W. S., M.D.....508 Dearborn Avenue, Chicago
Churchill, F. S., M.D.....460 Dearborn Avenue, Chicago
Cotton, A. C., M.D.....1485 Jackson Boulevard, Chicago
Crandall, Floyd M., M.D.....113 West Ninety-fifth Street, New York
Davis, Edward P., M.D.....250 South Twenty-first Street, Philadelphia
Dorning, John, M.D.....111 West Forty-eighth Street, New York
Edsall, D. L., M.D.....Philadelphia, Pa.
Forchheimer, F., M.D.....Fourth and Sycamore Streets, Cincinnati
Freeman, Rowland G., M.D.....205 West Fifty-seventh Street, New York
Graham, E. E., M.D.....1713 Spruce Street, Philadelphia
Griffith, J. P. Crozer, M.D.....123 South Eighteenth Street, Philadelphia
Hamill, S. McC., M.D.....1822 Spruce Street, Philadelphia
Holt, L. Emmett, M.D.....14 West 55th Street, New York
Huber, F., M.D.....209 East 17th Street, New York
Jackson, Henry, M.D.....309 Marlborough Street, Boston

JACOBI, A., M.D. ..... 110 West Thirty-Fourth Street, New York
JENNINGS, CHARLES G., M.D. ..... 457 Jefferson Avenue, Detroit
KERLEY, CHARLES G., M.D. ..... 113 West Eighty-third Street, New York
KOPLIK, HENRY, M.D. ..... 66 East Fifty-eighth Street, New York
LAFLEUR, HENRI A., M.D. ..... 58 University Street, Montreal
LOCKWOOD, WM. F., M.D ..... 8 Eager Street, Baltimore
MARTIN, C. F., M.D. ..... 33 Durocher Street, Montreal
MILLER, D. J. MILTON, M.D. ..... 345 South Eighteenth Street, Philadelphia
MORRILL, F. GORDON, M.D. ..... 181 Beacon Street, Boston
MORSE, J. LOVETT, M.D ..... 70 Bay State Road, Boston
NORTHRUP, WILLIAM P., M.D. ..... 57 East Seventy-ninth Street, New York
OSLER, WILLIAM, M.D. ..... 1 West Franklin Street, Baltimore
PACKARD, FRED'K A., M.D. ..... 110 South Eighteenth, Philadelphia
PUTNAM, CHARLES P., M.D. ..... 63 Marlborough Street, Boston
RACHFORD, B. K., M.D. ..... 323 Broadway, Cincinnati
ROTCH, T. M., M.D. ..... 197 Commonwealth Avenue, Boston
SAUNDERS, E. W., M.D. ..... 1635 S. Grand Avenue, St Louis, Mo.
SEIBERT, A , M.D. ..... 114 East Fifty-seventh Street, New York
SNOW, IRVING M., M.D. ..... 476 Franklin Street, Buffalo
STARR, LOUIS, M D ..... 1818 South Rittenhouse Square, Philadelphia
STENGEL, ALFRED, M.D. ..... 1811 Spruce Street, Philadelphia
TOWNSEND, CHARLES W., M.D. ..... 76 Marlborough Street, Boston
WENTWORTH, A. H., M D ..... 294 Marlborough Street, Boston
WEST, J. PARK, M.D. ..... 3608 Belmont Street, Bellaire, Ohio
WESTCOTT, THOMPSON S., M.D. ..... 1833 Spruce Street, Philadelphia
WILLIAMS, HAROLD, M.D. ..... 528 Beacon Street, Boston
WILSON, J. C., M.D. ..... 1437 Walnut Street, Philadelphia
WINTERS, J. E., M.D. ..... 25 West Thirty-seventh Street, New York
YALE, LEROY M., M.D. ..... 432 Madison Avenue, New York

Deceased.

JOHN A. JEFFRIES, M.D.,
Born, September 2, 1859,
Died, March 26, 1892.

THOMAS F. SHERMAN, M.D.,
Born, March 17, 1856,
Died, September 26, 1893.

JOHN M. KEATING, M.D.,
Born, April 20, 1852,
Died, November 17, 1893.

CHARLES WARRINGTON EARLE, M.D.,
Born, 1845,
Died, November 19, 1893.

J. LEWIS SMITH, M.D.,
Born October 15, 1827,
Died, January 7, 1898.

JOHN HENRY FRUITNIGHT, M.D.,
Born, November 9, 1851,
Died, December 18, 1900.

FOURTEENTH ANNUAL SESSION OF THE AMERICAN PEDIATRIC SOCIETY AT BOSTON, MASSACHUSETTS, MAY TWENTY-SIXTH, TWENTY-SEVENTH, AND TWENTY-EIGHTH, MDCCCCII

OFFICERS OF THE AMERICAN PEDIATRIC SOCIETY FOR MDCCCCII

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F. Forchheimer, M. D.
J. C. Wilson, M. D.
Floyd M. Crandall, M. D.
Thomas Morgan Rotch, M. D.

MONDAY, MAY TWENTY-SIXTH

MORNING SESSION, 10 A.M. to 12.30 P.M., at the Somerset Hotel, Commonwealth Avenue and Charlesgate East. Open to the profession in general.

I. THE PRESIDENT'S ADDRESS. *W. S. Christopher, M.D., Chicago.*

II. INTUSSUSCEPTION: CLINICAL REMARKS. *F. Huber, M.D., New York;* SURGICAL COMMENTS BY *John F. Erdman, M.D., New York.*

III. HEALED SEPTIC ENDOCARDITIS. *Samuel S. Adams, M.D., Washington, D.C.*

At 12.45 P.M., after the meeting, by invitation of the Automobile Club, the members of the Society will be taken through Cambridge to the Country Club, where lunch will be served at 2 P.M.

AFTERNOON SESSION. After lunch an Afternoon Session will be held from 3 to 5 P.M. and the following papers will be read:

IV. A CASE OF CHONDRODYSTROPHY FŒTALIS. *John Lovett Morse, M.D., Boston.*

V. PILOCARPIN IN SCARLET FEVER AND DIPHTHERIA. *E. W. Saunders, M.D., St. Louis.*

VI. THE MANAGEMENT OF RHEUMATIC CHILDREN. *Floyd M. Crandall, M.D., New York.*

Members will be taken back to the Somerset Hotel, where the Society will dine by invitation as guests at 6.30 P.M.

EVENING SESSION. There will be an Evening Session, open to the profession in general, at the Medical Library on the Fenway at 8.30 P.M. The following papers will be read:

VII. TYPHOID FEVER IN CHILDREN UNDER TWO AND A HALF YEARS OF AGE. *J. P. Crozier Griffith, M.D., Philadelphia,* and *Maurice Ostheimer, M.D., Philadelphia.*

VIII. TYPHOIDAL APPENDICITIS IN CHILDREN. *A. Seibert, M.D., New York.*

TUESDAY, MAY TWENTY-SEVENTH

MORNING SESSION, 9 to 12 A.M., at the Medical Library on the Fenway. Open to the profession in general.

IX. (*a*) REPORT OF A CASE OF EXTREME ENLARGEMENT OF THE SPLEEN WITH ANÆMIA; AUTOPSY. (*b*) A CASE OF VENOUS THROMBOSIS RESULTING IN EXTENSIVE CEREBRAL HEMORRHAGE IN AN INFANT FIFTEEN DAYS OLD; FUSION OF THE KIDNEYS. *Samuel McC. Hamill, M.D., Philadelphia.*

X. PRESENTATION OF CASES OF TUBERCULAR PERITONITIS. *Thomas Morgan Rotch, M.D., Boston.*

XI. HÆMOGLOBINURIA. *A. Jacobi, M.D., New York.*

XII. (*a*) THE USE OF THE TERM ENANTHEM. (*b*) SOME REMOTE DISEASES ARISING FROM TONSILLAR INFECTION. *F. Forchheimer, M.D., Cincinnati.*

XIII. (*a*) CLINICAL OBSERVATIONS ON THE MANAGEMENT OF CIRCULATORY FAILURE IN ACUTE INFECTIOUS DISEASE. (*b*) CHRONIC PARENCHYMATOUS NEPHRITIS IN A CHILD TREATED BY RENAL DECAPSULATION (EDEBOHL'S OPERATION). (*c*) SPECIMEN OF A LARGE THYMUS GLAND. (SUDDEN DEATH.) *Augustus Caillé, M.D., New York.*

BUSINESS MEETING. The Society will lunch at 197 Commonwealth Avenue at 1.30 P.M. After lunch there will be a Business Meeting and the following papers will be read:

XIV. TWO CASES OF UMBILICAL FISTULÆ DUE TO TUBERCULAR PERITONITIS. *George N. Acker, M.D., Washington, D.C.*

XV. EFFECTS OF TIGHT DIAPERS (illustrated). *A. C. Cotton, M.D., Chicago.*

By invitation the Society will as guests dine at the University Club, 270 Beacon Street, at 7 P.M., where tickets will be given to the members to adjourn after the dinner to the "Pop" Concert.

WEDNESDAY, MAY TWENTY-EIGHTH

THE members will be taken to the Eastern Yacht Club, where there will be a Morning Session, and where lunch will be served at 1 P.M. The following papers will be read:

XVI. DIPHTHERIA WITH AND WITHOUT ANTITOXIN. *Charles G. Kerley, M.D., New York.*

Following Dr. Kerley's paper *John H. McCollom, M.D., Boston,* will make some remarks on INTUBATION IN DIPHTHERIA.

XVII. TETANY OF THE TYPE CALLED PSEUDO-TETANUS, WITH AN ILLUSTRATIVE CASE OF DIPHTHERIA WITH PERSISTENT TRISMUS AND OPISTHOTONOS. *Irving M. Snow, M.D., Buffalo.*

AFTERNOON SESSION. After lunch the members will be taken by boat to the Misery Island Club, where an Afternoon Session will be held and the following papers will be read:

XVIII. THE MANAGEMENT OF THE PERCENTAGES OF FAT IN THE FEEDING OF DIFFICULT CASES IN INFANTS. *Thompson S. Westcott, M.D., Philadelphia.*

XIX. (*a*) RECENT INVESTIGATIONS UPON THE PROTEIDS OF MILK. (*b*) LOCAL VARIATIONS IN THE MORTALITY FROM SUMMER DIARRHŒA. *Henry D. Chapin, M.D., New York.*

XX. (*a*) A CASE OF AUTO-INTOXICATION WITH UNUSUAL URINARY FINDINGS. (*b*) NOTE ON THE TEMPERATURE CURVE IN ACUTE CROUPOUS PNEUMONIA. *C. G. Jennings, M.D., Detroit.*

XXI. REPORT OF A CASE OF PNEUMONIA. *Walter Lester Carr, M.D., New York.*

By invitation the Society will as guests dine at the Club in time to return to Boston, arriving at 10.33 P.M.

SAMUEL S. ADAMS, *Secretary,*
1 DUPONT CIRCLE, WASHINGTON, D.C.

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MINUTES OF THE FOURTEENTH ANNUAL MEETING OF THE AMERICAN PEDIATRIC SOCIETY.

Held at Boston, Mass., May 26, 27, and 28, 1902.

The meeting was called to order by the president, Dr. W. S. Christopher, of Chicago. The minutes of the thirteenth annual meeting were approved as published in ARCHIVES OF PEDIATRICS.

The following members were present: G. N. Acker, M.D., Washington; S. S. Adams, M.D., Washington; A. D. Blackader, M.D., Montreal; W. D. Booker, M.D., Baltimore; E. M. Buckingham, M.D., Boston; A. Caillé, M.D., New York; W. L. Carr, M.D., New York; H. D. Chapin, M.D., New York; W. S. Christopher, M.D., Chicago; A. C. Cotton, M.D., Chicago; F. M. Crandall, M.D., New York; D. L. Edsall, M.D., Philadelphia; F. Forchheimer, M.D., Cincinnati; R. G. Freeman, M.D., New York; E. E. Graham, M.D., Philadelphia; J. P. C. Griffith, M.D., Philadelphia; S. McC. Hamill, M.D., Philadelphia; L. E. Holt, M.D., New York; F. Huber, M.D., New York; H. Jackson, M.D., Boston; A. Jacobi, M.D., New York; C. G. Jennings, M.D., Detroit; C. G. Kerley, M.D., New York; H. Koplik, M.D., New York; C. F. Martin, M.D., Montreal; D. J. M. Miller, M.D., Philadelphia; F. G. Morrill, M.D., Boston; F. A. Packard, M.D., Philadelphia; C. P. Putnam, M.D., Boston; T. M. Rotch, M.D., Boston; A. Seibert, M.D., New York; I. M. Snow, M.D., Buffalo; C. W. Townsend, M.D., Boston; A. H. Wentworth, M.D., Boston; J. P. West, M.D., Bellaire; J. C. Wilson, M.D., Philadelphia; L. M. Yale, M.D., New York.

FIRST SESSION.—MAY 26.

Held at the Somerset Hotel.

The annual address of the president, entitled "Development, the Keynote of Pediatrics," was read by Dr. W. S. Christopher, of Chicago.

Dr. F. Huber, of New York, read a paper entitled "Intussusception; Clinical Remarks," and also one by Dr. J. F. Erdmann, of New York, entitled "Surgical Comments."

Discussion by Drs. Caillé, Packard, Jacobi, Putnam, Griffith, Chapin, Adams, Christopher, Blackader, Rotch and Huber.

Dr. S. S. Adams, of Washington, read a paper entitled "Healed Septic Endocarditis."

Discussion by Drs. Crandall, Wilson, Miller, Jacobi, Seibert and Adams.

SECOND SESSION.—MAY 26.

Held at the Country Club.

Dr. J. L. Morse, of Boston, read a paper entitled "A Case of Chondrodystrophy Fetalis," and exhibited photographs.

Discussion by Drs. Jacobi, Griffith, Holt, Christopher and Morse.

Dr. F. M. Crandall, of New York, read a paper entitled "The Management of Rheumatic Children."

Discussion postponed until evening.

THIRD SESSION.—MAY 26.

Held at the Medical Library.

Discussion of Dr. Crandall's paper by Drs. Chapin, Jacobi, Carr, Packard, Blackader, Koplik, Wilson and Crandall.

Dr. J. P. C. Griffith, of Philadelphia, read a paper entitled "Typhoid Fever in Children under Two and a Half Years of Age," by himself and Dr. M. Ostheimer.

Discussion by Drs. Christopher, Morse, Holt, Seibert, Koplik, Freeman, Jacobi, Miller, Packard, West, Adams, Wilson, Rotch, Kerley, Blackader and Griffith.

FIRST SESSION.—MAY 27.

Held at the Medical Library.

Dr. A. Seibert, of New York, read a paper entitled "Typhoidal Appendicitis in Children."

Discussion postponed.

Dr. T. M. Rotch, of Boston, read a paper entitled "Presentation of Cases of Tuberculous Peritonitis" and showed patients.

Discussion by Drs. Adams, Acker, Forchheimer, Koplik, Chapin, Caillé, Cotton, Jacobi, Miller, Seibert and Rotch.

Dr. F. Forchheimer, of Cincinnati, read papers entitled (a) "The Use of the Term Enanthem" and (b) "Some Remote Diseases Arising from Tonsilar Infection."

Discussion of the first paper by Drs. Caillé, Griffith, Wilson, Rotch, Seibert, Christopher and Forchheimer.

Discussion of the second paper by Drs. Jacobi, Freeman, Christopher, Jennings and Forchheimer.

Dr. S. McC. Hamill, of Philadelphia, read papers entitled (a) "Report of a Case of Extreme Enlargement of the Spleen with Anemia; Autopsy" and (b) "A Case of Sinus Thrombosis Resulting in Extensive Cerebral Hemorrhage in an Infant Fifteen Days Old; Fusion of the Kidneys."

Discussion of the first paper by Drs. Wentworth, Martin, Christopher and Hamill.

SECOND SESSION.—MAY 27.

Held at 197 Commonwealth Avenue.

Dr. A. Caillé, of New York, read papers entitled (a) "Clinical Observations on the Management of Circulatory Failure in Acute Infectious Disease," with illustrations; (b) "Chronic Parenchymatous Nephritis in a Child Treated by Renal Decapsulation (Edebohls' Operation)"; and (c) "Specimen of a Large Thymus Gland (Sudden Death)" with an exhibition of specimen.

Discussion of the first paper by Drs. Acker, Blackader, Kerley, Seibert, Jacobi, Cotton, Adams and Caillé.

Discussion of the third paper by Drs. Jacobi, Putnam and Caillé.

Dr. A. C. Cotton, of Chicago, read a paper entitled "Effects of Tight Diapers," and showed photographs and specimens.

Discussion by Dr. Rotch.

FIRST SESSION.—MAY 28.

Held at Eastern Yacht Club.

Dr. G. N. Acker, of Washington, read a paper entitled "Two Cases of Umbilical Fistula due to Tuberculous Peritonitis" and showed photographs.

Dr. C. G. Kerley, of New York, read a paper on "Diphtheria With and Without Antitoxin."

Dr. J. H. McCollom, of Boston, made "Some Remarks on Intubation in Diphtheria."

Discussion of both papers by Drs. Chapin, Caillé, Jennings, Buckingham, Cotton, Crandall, Adams, McCollom and Kerley.

Dr. I. M. Snow, of Buffalo, read a paper entitled "Tetany of the Type Called Pseudotetanus With an Illustrative Case of Diphtheria with Persistent Trismus and Opisthotonos."

Discussion by Drs. Jacobi, Koplik, Holt, Huber, Kerley, Griffith and Snow.

Dr. T. S. Westcott, of Philadelphia, read a paper entitled "The Management of the Percentages of Fat in the Feeding of Difficult Cases in Infants."

Dr. H. D. Chapin, of New York, read papers entitled (a) "Recent Investigations Upon the Proteids of Milk" and (b) "Local Variations in the Mortality from Summer Diarrhea."

Discussion postponed until afternoon.

SECOND SESSION.—MAY 28.

Held at Misery Island Club.

Discussion of Dr. Westcott's paper and of Dr. Chapin's first paper by Drs. Holt, Buckingham, Miller, Cotton, Koplik, Townsend, Edsall, Freeman, Wentworth, Griffith, Westcott and Chapin.

Discussion on Dr. Seibert's paper on "Typhoidal Appendicitis" postponed from May 27, by Drs. Caillé and Seibert.

Discussion on Dr. Chapin's second paper by Drs. Seibert, Adams, Cotton and Chapin.

Dr. C. G. Jennings, of Detroit, read papers entitled (a) "A Case of Autointoxication with Unusual Urinary Findings" and (b) "Notes on the Temperature Curve in Acute Croupous Pneumonia," with charts and drawings.

Discussion of the first paper by Drs. Edsall and Christopher, and of the second by Dr. Morrill.

Dr. W. L. Carr, of New York, read a paper entitled "Report of a Case of Pneumonia."

Discussion by Drs. Adams and Carr.

The following papers were read by title:

Dr. E. W. Saunders, of St. Louis, "Pilocarpin in Scarlet Fever and Diphtheria."

Dr. A. Jacobi, of New York, "Hemoglobinuria."

EXECUTIVE SESSION.—MAY 27.

The report of the Council was presented by its chairman, Dr. Blackader, and accepted.

On nomination of the Council the following officers were elected for the ensuing year:

President, - -	J. P. CROZER GRIFFITH, M.D., Philadelphia.
First Vice-President,	HENRY D. CHAPIN, M.D., New York.
Second Vice-Pres.,	FRANK S. CHURCHILL, M.D., Chicago.
Secretary, - -	SAMUEL S. ADAMS, M.D., Washington.
Treasurer, - -	J. PARK WEST, M.D., Bellaire, Ohio.
Recorder and Editor,	WALTER LESTER CARR, M.D., New York.
Member of the Council,	F. A. PACKARD, M.D., Philadelphia.

Elected to membership, Alfred Hand, Jr., M.D., and W. Reynolds Wilson, Jr., M.D., of Philadelphia, and Henry L. K. Shaw, M.D., of Albany.

Place and time of meeting—*Washington, D. C., May 12, 13 and 14, 1903.*

Assessment for ensuing year—*Five dollars.*

The president was authorized to appoint delegates to the Fourteenth International Medical Congress.

The offer of ARCHIVES OF PEDIATRICS to print the Transactions was accepted.

An appropriation of $25 was made for American Committee of the Fourteenth International Medical Congress.

An appropriation of $200 was granted for the indexing of the Transactions and $100 for publishing papers not inserted in Volume XIII. of the Transactions.

WALTER LESTER CARR, M.D.

Recorder.

PRESENTATION OF A LOVING CUP TO DR. ROTCH.

At the close of the Boston meeting a loving cup was presented to Dr. T. M. Rotch.

The presentation was accompanied by the following graceful remarks by Dr. Leroy M. Yale, of New York:

"I have been asked to say a few words in behalf of those members of the Society who have come from other cities. I am sure that they will first wish me to try to express to you how much they enjoyed their visit to Boston. Those of us who have been here before, of course, knew what a pleasure was before them. For myself, I always like to come to Boston, for I acquired the Boston habit very early in life. This was the first considerable city I ever saw, and I have never since seen one so splendid as it then seemed to my country-bred eyes.

"But it was quite another Boston, small in area but high in spirit. Think of it—'The Autocrat' and 'Hiawatha' had not yet charmed it. Its blood had not yet boiled at the extradition of Burns. The hero whose splendid memorial graces the Common was still a schoolboy. Even now I like to orientate myself by beginning at Boston Stone, at Copp's Hill or the Old North Church, before I wander out to the Public Garden, where the little waves beating upon its rip-rap edges seemed there to end the town, just where a new city—a new world almost—now begins.

"What impresses a visitor from another city is that all this change and increase is not mere expansion, but growth. Many towns seem to spread indiscriminately, like a neoplasm, but Boston has developed structurally. With a foresight unexampled, so far as I know, in this country, its growth has been planned for in such a way that it may go on increasing normally, healthfully and beautifully for generations. I have thought that

the original restricted domain of Boston has proved to be an advantage. It promoted unity of action, and when the town felt the need of more land it put it just where it wanted it, instead of taking any ready-made adjacency.

"But with all this splendid material growth, I find some things unchanged. Boston still thinks as of old. Still she responds as quickly as ever to the touch of an idea; still is the spirit of light and leading in her; still we feel *ex oriente lux;* still is her hospitality as rich as ever. I felt it when the driver of the Charlestown 'Hourly' picked me up—an eight-year-old boy, who had bankrupted himself at Martin L. Bradford's tackle shop beside the Old South Church; took my pole on top of the bus and gave me a free ride on the footboard behind. I felt it when the men who were then giving Boston her medical name treated me, a young graduate, as if I were 'somebody in particular.' Never has it been more evident than now, when you have not only shown us hospitality, but have given us a lesson in the art of showing it. So gracefully have you done it that we have taken with pleasure an amount of attention which, when we read it in the program, seemed appalling.

"Dr. Rotch, we desire to offer you this loving cup as a token of our regard. We offer it to you not only as our host; I should like to tell you some of the other reasons. But if there is anything that is hard to speak and hard to hear it is praise to one's face. It will please you, I am sure, to know that the suggestion to give it met with eager, immediate and unanimous response. It will strengthen your hands to know that of those here gathered from this wide country of ours, there is not one who does not feel that he does his daily work with greater ease and with greater certainty because of your labors.

"We have filled this cup with a 'modification' after an old prescription, a native of Northeastern Massachusetts. We trust that its 'percentages' are judicious. As we hand it to you, and as it passes from lip to lip, we pledge you as our comrade, as our master and as our friend."

DEVELOPMENT, THE KEYNOTE OF PEDIATRICS.

PRESIDENTIAL ADDRESS.

BY W. S. CHRISTOPHER, M.D.

Chicago.

While thanking my colleagues for the honor they have seen fit to confer upon me in presenting me the presidency of this organization, I wish also to congratulate them upon the line of work in which the Society is engaged, for I am firmly convinced that no other department of medicine has to do with more fundamental biologic truths, has as great possibilities for usefulness, has as much unexplored domain awaiting research, as this department of pediatrics, and I aim here to present the reasons for the faith which is within me.

With the question whether pediatrics is a specialty I have no concern, regarding such discussions as fruitless. But with its relation to general medicine and with its practice and study I am much interested. It is clearly a part of general medicine; this is a matter of definition. But general medicine as it has been taught, and as it has been practiced, has been singularly free from any active interest in the problems of pediatrics. I know of no textbook on so-called general medicine which evinces the slightest conception of the fundamental philosophy of pediatrics. The fact is that that which has posed as general medicine is a very thorough presentation of the special manifestations of the diseases of adult life, with some occasional reference to disease as it shows itself in children, but lacking entirely in any systematic presentation of the forces at work which demand for the diseases of childhood special study, and which have such important etiologic relations to adult pathology.

It is certainly true that scarlet fever is in the main the same whether it occur at two years of age or at forty; so is diphtheria; rather greater differences due to age, are found in typhoid fever. But it is not these differences, the importance of a knowledge of

which is not denied, that constitute the framework of pediatrics. Indeed, all the infectious diseases of childhood are only accidentally children's diseases, and are largely dependent upon early opportunity of infection.

I do not wish to be considered as criticising the magnificent work which has been done in general medicine, but only to be considered as calling attention to certain important omissions for which good and sufficient reasons exist.

The one feature which characterizes pediatrics and is its framework is development. But it is enough. One-third of the life of the individual who completes his threescore years and ten is devoted to this period of development. Then comes the long stretch of physical standstill to be followed by the short one of degeneration and decline. Death reaches us as the culmination of the accumulated insults to which the body has been subjected during its career. It occurs when the attacking force equals the resisting force. The physical care of the adult consists in diminishing and relieving the effects of these insults and some warning advice as how best to avoid them. The physical care of the child includes not only as much, but introduces also a positive constructive element. It aims to so control the environment of the developing individual, that the resisting force of the adult shall be made to reach the greatest degree possible. In other words, it aims to make of the given child the strongest possible adult. Pediatrics, therefore, is preventive medicine of the highest order, and is only possible because of the existence of the developmental period of human life, and because this development can be acted upon, and acted upon strongly, by environment, and either advantageously or disadvantageously.

It so happens that of all animals, man has the longest period of development, not only as to the actual time involved (except as to the elephant), but also proportionately as to the total natural duration of life. John Fiske has shown that this long period of plasticity is the factor which has permitted man to rise so far superior to the other animals. A brief presentation of Fiske's views would seem to be in order, and as I have presented them elsewhere,* I shall here merely quote therefrom:

"The life of the codfish is a simple one. Its acts are mostly concerned with the securing of food and the avoidance of danger.

* *Chicago Record-Herald's* Current Topics Club, May 19 1901.

These acts are few in kind and require for their performance only a very slight intelligence. Its experiences, while numerous enough, are so much of a kind that practically they require only the monotonous repetition of the same few acts. So few are these acts and so limited the nervous connections necessary to their proper performance, that they become established by heredity, and the young codfish enters upon its life capable of performing all of them about as well as its ancestors. It has little to learn from experience. It requires no education. It has no infancy.

"Consider, on the other hand, an expert pianist. He acquires the skill to read his music, slowly; he acquires the skill to perform it, stumblingly, but finally he will perform the most difficult music at sight, and do it easily. Slowly has he developed the nervous connections necessary to these acts. They were not developed at birth. Analogous has been the development of the great artist, the great poet, the great warrior, the great mathematician.

"To a less degree, but in the same manner and by similar mechanism, has the mediocre man developed his power to do what he does. Even the lowest man in the race develops after birth his most important voluntary motor functions. The power to do, whether by muscle or by brain, is in man almost exclusively a post-natal acquirement. Between the highest and the lowest animals a great diversity of post-natal acquirement exists. . . .

"The young puppy is quite helpless at birth. But his infancy is short, and he soon crystallizes into an adult dog. Yet short as is the infancy of this species, dog-fanciers have taken advantage of it and by careful training and selection, have developed many interesting varieties of this animal. These men fully recognize that the period of infancy is that of plasticity, for they say, 'It is hard to teach an old dog new tricks.' The acts of the adult animal of this species are so simple that a short infancy is all that is necessary to gain the experience and adjust the nervous connections required for the performance of the adult acts. . . .

"Wallace relates in his 'Malay Archipelago' his observations of two infant quadrumana, which he was fortunate enough to have in his possession at the same time. One was the infant of a hair-lip monkey, Maccacus cynomolgus, an ordinary animal such as organ-grinders use; and the other was an infant mias or orang-outang, the highest and most intelligent of the man-like apes. He says: 'It was curious to observe the different actions of these two

animals, which could not have differed much in age. The mias (orang-outang), like a very young baby, lying on its back quite helpless, rolling lazily from side to side, stretching out all four hands into the air wishing to grasp something, but hardly able to guide its fingers to any definite object, and, when dissatisfied, opening wide its almost toothless mouth, and expressing its wants by a most infantine scream. The little monkey, on the other hand, in constant motion, running and jumping about wherever it pleased, examining everything around it, seizing hold of the smallest objects with the greatest precision, balancing itself on the edge of the box or running up a post, and helping itself to anything eatable that came its way. There could hardly be a greater contrast, and the baby mias looked more baby-like by the comparison.'

"Of the various animals reviewed, it is to be noted that the higher the intelligence of the adult animal, and the more complex its life activities, the longer is its infancy or period of development. Each increase in brain size is accompanied by increase in intelligence, and, generally speaking, this increase in intelligence is accompanied by greater complexity of the life of the individual; that is, there is a greater variety of experiences and a more numerous development of faculties, and hence a greater number of nerve connections to be established to direct and control these faculties and their resulting activities. Finally, there comes a time when the necessary nerve connections can not all be established before birth, but must be partly established by education after birth. Thus is infancy established, for any animal requiring post-natal development, which should happen not to have a period of infancy, would cease to exist. Even in the human family it is far from uncommon to meet instances where both pre-natal and post-natal development are from some cause or other hindered, and where in consequence the resulting individual would cease to exist, were it not for the fostering care which highly organized society gives him."

Thus it seems that our species has been dependent for its evolution to its present high status upon its prolonged period of plasticity, which has permitted relatively rapid differentiation of offspring from parents. But this refers to phylogeny; we are concerned with ontogeny. Racial development has come to pass through the slow processes of natural selection, but in the de-

velopment of the individual, intelligent and more direct means can be brought to bear, and the wonderful opportunity is the prolonged plasticity of the species.

To take advantage of this opportunity, inasmuch as development is primarily determined by heredity, it is necessary to know first the course of natural development, and next the pathological conditions resulting from deviations from that course, together with the causes of these deviations. Finally, the environmental factors must be classified, and their influence in maintaining normal development and in permitting and causing deviations therefrom, determined. These aspects have been studied conjointly, and while not so much is known as is desirable, yet there exists a body of knowledge sufficient to make an efficient, constructive pediatrics.

Much work has been done in the mensuration of anatomical features of the body with a view to determining their laws of growth, and it is gratifying, as well as surprising, to find how practical this information is in its application to the management of individual children. The study of development from the anatomical standpoint is at bottom a very simple matter, requiring principally the opportunity. It involves, however, accuracy of measurement and data from large numbers of individuals. Such data can be gathered in part by each one of us, and the collective result be made valuable.

Functional development has not been subjected to so searching an inquiry, except in the case of the nervous system, which presents rare opportunities for quantitative investigation, of which advantage has been taken by the physiologists whose results we would do well to make our own. The functions of the kidneys have been fairly well studied, and through the urine, some attempts, although very inadequate ones, have been made to study developmental metabolism. Of remaining organs, a knowledge of whose functional development would be most serviceable, the most important are the liver and the ductless glands. However, in view of the insufficiency and uncertainty of present information regarding the specific functions of these organs, it seems too much to hope that the near future will afford any adequate insight into the natural history of the development of these functions. While we are waiting for the physiologists to supply us with this natural history of the develop-

ment of the functions of the several organs of the body, much can be accomplished by clinical methods of observation. At least four diseases of autotoxic character are to be met with in adult life. These are gout, in all of its numerous manifestations, diabetes mellitus, Grave's disease, and Addison's disease. All of these have their manifestations not only in childhood, but also in infancy. Each represents a certain perversion of the chemism of the body. These perversions of chemism are found much more commonly in the undeveloped infant than in the adult, and it would seem that, in many instances at least, the adult manifestations are but the persistence and amplification of the infantile and perhaps fetal types, and in gout at least, there is distinct reversion to functions of lower forms of life. Moreover in the infant these perversions of chemism are found intimately intermixed, but generally as development goes on, separation occurs, and it is unusual to find more than one form persisting in the adult. Possibly, also, death has eliminated some of the individuals who have been most heavily handicapped. This is the chemical analogy of Cohnheim's anatomical theory. It is in these conditions of perverted chemism of infancy that we find many of the difficulties of infant feeding. It is to them that we must attribute such phenomena as the toxicity of milk in every form to some infants; the toxicity of raw milk and the healthfulness of boiled or condensed milk to others; the utility of beef juice at so early an age as two weeks to some babies, and its toxicity to others for so long as the first three years of life. It is to the type of chemism that we must look for our explanation of the tendencies of infants and children to various degrees of obesity, and for the existence in them of such varying assimilative factors. It is perverted chemism, with its frequent resulting autotoxemia, which we must consider in many of the skin diseases of children, in their anemias, in their tendencies to recurrence to certain forms of disorder. In short it is the type of chemism of the body which determines the diathesis. And all these are but features of development.

Of the environmental factors concerned in the life of the child, nutrition and infection stand preeminent, with its physical and mental activities making a close second. Rather less important is the means of protection from the elements, including clothing, dwelling, and climate and other geographical

considerations. By no means to be omitted is the matter of discipline. Nutrition is a very comprehensive term. Not only does it include the matter of food stuffs, their digestion, absorption and assimilation, but also such profound considerations as the nature of the normal constructive metabolism, the nature and causes of hyperconstructive metabolism, and destructive metabolism, together with the numerous autotoxemias of infancy and childhood. Of the relations of nutrition to infections, only the most superficial knowledge is yet at hand. How desirable it would be to know the nutritional factors which determine the overwhelming frequency of scarlet fever in the second two years of life.

But even a superficial consideration of these environmental factors would lead farther than would be profitable on such an occasion, and I close this portion of my address, offering the Society for a motto John Fiske's saying: "It is babyhood that has made man what he is."

I beg now to call the attention of the Society to two matters which I feel that I have the privilege of presenting. There is a movement on foot to induce the Federal Government to establish at Washington, in the Department of the Interior, a laboratory for the study of criminal, pauper, and defective classes. Such a laboratory must collect, and indeed it is one of the avowed intentions to collect, much data bearing upon the development of the child. We are asked to favor the movement, and I strongly recommend the passage of a suitable resolution to that end.

It is certain that the Society is desirous of attaining as high a degree of usefulness as possible, and to that end to extend its influence as far as possible to the furtherance of the study of pediatrics. It owes this high duty to itself and to the profession. As an organization, it is necessarily judged by its collective work as given in the bound volumes of its Transactions. It is therefore necessary that every proper effort should be made to secure from members their best productions for the Transactions of the Society. While the bound volumes of the Transactions must ultimately determine the standing and influence of the Society as a whole, they do not present a good field for the general distribution of the Society's work, and this must be sought by preliminary publication in regular journals. In the selection of the journal for original publication, the widest freedom should be allowed to the

individual member. The present plan of publishing all papers read before the Society in one journal of the Society's selection, has a distinct tendency to keep away such papers as members may wish to publish elsewhere, and thus deprive the Transactions of some of the members' work. Others indeed may read their papers before the Society, and then deliberately violate the rule of the Society as to place of publication, in this way again depriving the Transactions of matter which belongs to them. I confess myself a sinner of this latter type. In every way we look at it we find the present arrangement is one which tends very directly to diminishing the value of our Transactions and thus diminishing the influence of the Society. I therefore recommend that the Society undertake the publication of its own Transactions.

INTUSSUSCEPTION—CLINICAL REMARKS.

BY FRANCIS HUBER, M.D.,

Visiting Physician to Gouverneur Hospital; Junior Visiting Physician, "Jacobi Ward," Roosevelt Hospital, New York.

WITH SURGICAL COMMENTS.

BY JOHN F. ERDMANN, M.D.,

Clinical Professor of Surgery, University and Bellevue Hospital Medical College, New York.

An early recognition of the lesion and a knowledge of the proper limitations of the methods by injections or inflation, are essential to the successful treatment of acute intussusception. The case reported below brings out strongly many interesting features and will serve as a text for further remarks.

HISTORY.—F. H., male, eight and one-half months of age; weight, nineteen pounds, general health good, was vaccinated about ten days previously. He was partly breast fed; several times daily he received small quantities of cow's milk boiled and diluted with cereal decoction; once daily a few ounces of broth; orange juice occasionally. No history of trauma or "dancing the baby."

March 21st.—He was taken with a sharp attack of diarrhea (seven stools in twenty-four hours containing undigested casein and some mucus: consistency rather soft). The intestinal disturbance was not attended with vomiting nor did the infant appear to suffer from any pain or discomfort, as he continued to be lively and playful.

March 22d, 8 A. M.—He had a large liquid stool, painless, coming with a gush, showing increased and vigorous peristalsis. At 10 A. M., was given his usual bath, nothing abnormal noticed in his disposition or looks, for he splashed and played as usual. Taken out in his carriage about 11.15, he quickly became restless, uneasy and fretful. This was unusual, as he generally slept when taken out for an airing. The restlessness increased so he was

taken home. No vomiting or further stool. At 12.30 he received an enema of warm salt water; this was followed by a small undigested stool with the escape of considerable gas. He then fell asleep to awake about 1.45 P. M. with severe abdominal pains, colicky in character, recurring in paroxysms of five to twenty minutes, with periods of rest in the intervals. Nausea followed by vomiting set in about two o'clock. At first the contents of the stomach were vomited, later thick tenacious mucus somewhat stained with bile appeared. The vomiting was projectile in character; the antiperistaltic movements distressing and painful. He would not nurse and was very restless. When seen again at 4 P. M., had just vomited a thick tenacious mucus with a faint odor of sulphuretted hydrogen. Expression of face changed. Evidently severely ill, the child presented an anxious appearance with pallor of surface and muscular relaxation; marked restlessness noted. No decided collapse. Vomited incessantly, each attempt distressing in the extreme.

PHYSICAL EXAMINATION revealed a soft, relaxed and somewhat retracted abdomen. On palpation a slight swelling or rather fulness was appreciated in the region of the transverse colon. The paroxysms of pain recurred with increasing frequency and the antiperistaltic movements of the stomach were distinctly visible through the abdominal walls when the child was exposed. No action of the bowels since the enema was given five hours previously. No tenesmus. A high rectal injection was now given, the hips being elevated. As the water escaped a bloody and mucous discharge amounting to about half an ounce, resembling red currant jelly, followed.

This was a positive confirmation of the existence of an acute intussusception. Drs. J. F. Erdmann and M. H. Silver were now requested to see the case in consultation, reaching the patient about 5.20 P. M. A rectal examination by Dr. Erdmann failed to reveal a tumor, nor could the fulness referred to above be made out at this examination. The case being an early one, it was concluded to make an attempt to reduce the intussusception by means of hydrostatic pressure with gentle manipulations of the abdomen under chloroform, and if unsuccessful to operate at once. Under narcosis, an elongated tumor could be distinctly seen occupying the position of the transverse colon.

A pressure of about five feet with gentle abdominal manipula-

tions by Dr. Silver, succeeded in causing a reduction or rather disappearance of the tumor. After the escape of the water no localized swelling could be detected upon careful palpation of the abdomen. Within a few moments, considerable mucus with numerous specks of blood appeared on the diaper. A detailed consideration of the case lead us to believe that only a partial reduction had taken place. In a number of successful cases of reduction by injection in my own experience, a fecal passage invariably followed the relief of the obstruction. In our case, in the absence of a fecal discharge, we were left in doubt. It was therefore thought best to perform an abdominal section, to clear up the case rather than to wait for further, perhaps unfavorable, symptoms.

THE OPERATION, including the removal of the appendix, for reasons given below, did not take longer than twelve minutes. Vomiting persisted until the following afternoon. During the night some offensive gas escaped. Twenty hours after the operation a liquid offensive stool with considerable foul smelling gas, was passed. The child was kept quiet with small doses of morphia and later on camphorated tincture of opium was administered. From this time on, with the exception of some temperature due to the secondary enteritis, and which latter gradually abated in about two weeks, no untoward symptom arose. The dressings were changed at the end of a week, the wound found to have healed primarily, and the sutures removed. An adhesive plaster dressing and flat gauze pad were continued for several weeks to prevent any yielding of the cicatrix. Up to the present the child is improving and no unpleasant manifestations have appeared.

Before taking up Dr. Erdmann's discussion of the surgical aspect, it may be well to review the points in our case. To recapitulate, an infant in apparent good health after a short attack of diarrhea becomes restless and refuses to nurse, is seized with recurring abdominal pains, paroxysmal in character, followed shortly by vomiting, the attacks increasing in frequency, becoming more and more distressing. The face becomes pale, assumes an anxious look, the eyes sunken. No decided collapse. A sense of fulness is noticed over the transverse colon upon palpation of the abdomen, though a distinct "elongated tumor" cannot be made out until chloroform is given an hour later. Bloody discharge observed after a high rectal enema. No rectal tumor discoverable.

Edmund Owen (*British Medical Journal,* September 7, 1901) gives the following summary of the symptoms of this lesion. (1) Sudden abdominal pain; (2) vomiting; (3) the passage with much straining of a stool containing mucus, blood and scanty liquid feces; (4) the presence of a tumor in the region of the ascending colon.

One or more of the symptoms characteristic of the disease may be absent or may not appear until late. In the discussion we must consider two factors; First, the character of the attack, whether acute, subacute or chronic, and second, the seat of the lesion, whether "enteric," ileocecal or colec. The urgency of the symptoms will depend upon the situation and the degree of the obstruction or strangulation. The more acute the case, the more outspoken are the symptoms and the greater the danger. In the subacute and chronic cases the prognosis is better and the symptoms are of a milder type.

The site of the invagination will influence the picture. The higher up the lesion the more marked are the gastric symptoms and pain. In the "enteric" variety, the least frequent form in children, a palpable tumor is the exception. The usual variety is the ileocecal, and our remarks are confined to this class.

Pain is a constant symptom; in fact it has been well said, "the characteristic colic of intussusception is a diagnostic sign of the greatest importance and it is from a recognition of its peculiar features that earlier diagnosis and earlier treatment is to be hoped." Sudden in its onset, of great intensity and attended with screaming spells and other evidences of acute suffering, it is, moreover, paroxysmal in character with shorter or longer intervals of relief. English authorities suggest that in every case of acute colic in infancy, the question of intussusception should be considered, and the examination be directed to its discovery.

Vomiting sets in early. It is persistent, uncontrollable and often projectile in character. The antiperistaltic movements of the stomach are plainly evident when the abdomen is exposed for inspection. At first the contents of the stomach are rejected, sooner or later, depending upon the acuteness of the case, it becomes bilious in character. Fecal vomiting is a rare event in infants. If the lesion be high up in the small intestine (enteric variety) a grumous material may be vomited, as occurred in a case observed quite recently.

The character of the rectal discharge is important. In acute cases, when invagination has occurred, the pressure upon the mesentery involved produces venous obstruction, thereby giving rise to edema and extravasation of blood in the intussusceptum, and even hemorrhage from the mucous membrane implicated in the process. At the outset one or more fecal movements are observed, followed by clear blood or bloody mucus resembling red currant jelly, the latter occurring particularly after a paroxysm of pain. This is characteristic, for, with the exception of twists and knots, no other form of obstruction is accompanied by such evacuations. In dysentery and rectal polypi the history is different.

Traumatism from the unskilled use of a hard nozzle in administering injections or in consequence of sudden unexpected movements on the part of the child may cause blood to appear. Now and then hard fecal masses will act in the same way. In scurvy and purpura blood may escape from the rectum, but the presence of other signs will clear up the case. Recently, for a few hours, some doubt existed in the case of a small boy who had severe abdominal pains with bloody discharges, and who at the same time had pains in various joints. A high rectal injection with thin starch water and the internal use of codein and sodium salicylate, after a few doses, cleared up the diagnosis. As the case progresses the color becomes darker, clots may pass and tenesmus appears. The rectal mucous membrane is everted through the relaxed sphincter. Finally, the tumor may be present at the anal orifice. Not infrequently after twenty-four hours, and at times earlier, the bloody and mucus discharges have a decided gangrenous odor.

Even before the appearance of a tumor, a rectal examination may be followed by a characteristic bloody flow. In the beginning the abdomen is soft, relaxed and possibly somewhat retracted. Palpation is not difficult. There may be a sense of resistance or slight fulness in the right iliac fossa without tenderness. As the case progresses a lump is felt. Eventually the characteristic "sausage-shaped mass," which becomes more pronounced during the colicky attacks, can be made out along the transverse colon. In some cases the tumor can be felt per rectum as early as ten to twelve hours from the onset of the symptoms.

Conjoined abdominal and rectal exploration, preferably under narcosis, may reveal a tumor not discoverable by simpler means.

In the case reported a prominent and early sign was the change in the general condition and facial expression. The extremities were cool, the face pallid, eyes sunken and extreme restlessness with muscular relaxation was noted. The degree of collapse varies. In some cases it is pronounced, the temperature may be subnormal, pulse accelerated and weak, etc. Profuse cold perspiration may be a prominent symptom.

My remarks have been limited to a consideration of the early signs. Later on, sepsis, peritonitis (local or general) or gangrene change the picture and render the prognosis more dangerous.

In several articles upon this topic, we are told that spontaneous reduction is possible.

In proof of this instances are cited of attacks of sudden paroxysmal pain, accompanied by stools containing blood and mucus, the attacks disappearing suddenly, enteritis continuing for a few days. Such a result cannot be expected in many instances. First, the swelling of the apex of the invaginated portions renders this mechanically impossible; secondly, the peristaltic efforts of the intussuscipiens are in an outward direction. Finally, in consequence of the lateral traction of the mesentery, the axis of the intussuscepted portion no longer corresponds with that of the intestines, thus the condition becomes more complicated and reduction is more and more difficult.

Injections and operative measures are rival methods of treatment. Each plan has its advocates. Progressive surgeons, particularly in hospital practice, favor early abdominal section. Injections, and under the term are included inflation of the bowels with air or distention by hydrostatic pressure (the latter considered safer), have been advised. First, for the purpose of reducing the invagination; second, with the object of lessening the size of the tumor and thereby limiting the field and simplifying the subsequent operation. Finally, in occasional cases the sufferings of the patient are lessened by the use of an enema prior to operation. At present the tendency is to limit the employment of injections to the early stages, that is, not later than twelve to twenty-four hours from the onset of the symptoms. It is recognized that the longer the condition has existed, the greater the dangers of

pressure from within. If not successful immediately, further attempts ought to be given up and abdominal section resorted to without delay.

Edmund Owen is very emphatic in his denunciations and writes as follows: "I deem it nothing less than a calamity that physicians every now and then manage to chase back an intussuscepted piece of bowel by using an enema."

Whether inflation by air or hydrostatic pressure be preferred, the following objections or disadvantages are apparent: First, it is impossible to gauge the amount of pressure. Second, except in the earliest cases it is not possible to exclude beginning gangrene, which, as shown by the results of operation, may take place very early. Third, the reduction is not always complete. The tumor may disappear, the symptoms abate, to recur again in a few hours. We therefore cannot be positive that with the reduction of the swelling, the entire mass has been reduced. There is always an element of doubt, consequently the procedure has been termed uncertain and dangerous, haphazard and therefore unscientific.

When disinvagination has been successful the relief is immediate. The anxious pallid look disappears and with it the evidence of collapse. The abdomen assumes a normal contour. In several instances in my own experience, fecal movements with the escape of gas, followed closely upon reduction. Now and then a rumbling noise is a favorable sign. Relapses are not uncommon.

As to subsequent treatment, the patient should be kept absolutely quiet with opium and an abdominal bandage, just tight enough to steady the bowels, must be applied.

A word as to the method of employing injections. They may be given with or without anesthesia. Though the former is preferable, the latter has this advantage, if the reduction is successful, the change in the expression of the face and relief of symptoms are noticed at once in a child not under the influence of chloroform. The patient becomes quiet at once, takes food easily and soon goes to sleep.

The following simple plan, advocated by Jacobi, may be followed: "The baby is turned on its belly, the hips are raised, the abdomen gently supported by a soft pillow. The mouth and nose, being the lowest part of the body, must be protected. The baby is then anesthetized with chloroform, and warm water is poured into the rectum with but little pressure, not from a height of

'fourteen feet.' This is important, for the intestine is no iron pipe, subject to the laws of hydrostatics only. The injection is frequently intermitted, while the anus is closed by the finger. At the same time the abdomen, in the direction from below upward, is gently kneaded and its contents moved about."

SURGICAL COMMENTS.

Very naturally the paramount question to be answered, when the surgeon is called, is that of the ultimate outcome, particularly is this so when the cases operated upon are in the very young.

In a paper, "Intussusception, with a Report of Three Additional Operative Cases," read before the Orange Mountain Medical Society, October, 1901 (*New York Medical Record,* 1902), this subject was covered quite thoroughly, and I gave in addition an abstract of all my previously operated cases. Since this paper was read I have operated upon six additional cases, including the one reported above. From a study of these cases I feel that I am in the position of one who can safely say that the operation for intussusception is accompanied by far less a mortality rate than that which occurs when hydrostatic pressure, inflation, etc., are practiced, and I am positive that surgical interference is the only treatment when a case has passed the twelve hour period of duration.

Unfortunately for all, the rate of mortality rapidly increases for every added twelve hours, and after the third day the rate of recovery is so small that one is discouraged when considering it in the surgical sense. I make it a rule when called to see a case of twelve hours' duration and over not to use enemata, etc., but to request the privilege of operating, as in all these cases the surgeon is only called after all mechanical methods for reduction have been practiced; furthermore, after a period of three to six hours' duration the congestion and edema may be sufficient, as a result of strangulation, to prevent any reduction even by surgical manipulations.

In several cases operated upon by me in which lacerations or ruptures of the serous coat have been found it has seemed to me that this condition was due to hyperdistention of the bowel by water or air rather than to the distention of the gut by the intussusception. The shock present in very many of these cases is

not a contraindication to operation but calls for more rapid work in reduction by surgical means. After the operation the child recovers rapidly from the shock, and either passes into a comfortable sleep or upon awakening from the anesthetic presents the picture of an apparently healthy child.

The operation is an extremely simple one, requiring asepsis and expedition more than anything else during the first twenty-four hours, and rarely, except in the long standing cases, is excision of the gut required. After the first twenty-four hours one is apt to meet with tears in the serosa, adhesions between the entering and the entered gut, preventing easy reduction, and finally, gangrene (either localized in spots or of considerable extent) calling for the various procedures allowable under the physical conditions found.

The position of the incision depends to a degree upon the presence of the tumor, *i.e.*, should a tumor be found in the region occupied by the descending colon or sigmoid, then a left rectus incision is far better for the proper manipulation of the gut, although I rarely find it necessary to open except through the right rectus.

This latter region is the one I usually select whether the tumor be present or not for the reason that the final portion of gut to be reduced is usually in the ileocecal region. Furthermore, all ordinary manipulations in any part of the abdomen can take place through an enlargement of this incision. The recti are selected in preference to the median line for the usual surgical reason of preventing herniation.

Personally I have never found it necessary to stitch the gut or mesentery, or mesocolon, to the parietes for the usually ascribed cause of long lax mesentery or mesocolon. I have always felt that sufficient temporary adhesions would form as a result of the congestion and edema of the gut that is finally extruded from the intussuscipiens.

In the case reported by Dr. Huber, several surgical points of interest were noted. Upon cessation of the use of the injections the abdomen was distended, evidently by some water remaining to such a degree as to render positive palpation useless. The child's condition of semishock still obtaining to a degree, no feces nor flatus being passed, it was decided to again anesthetize the patient and do a laparotomy.

Under chloroform anesthesia a vague sense of tumefaction was observed upon palpation in the right iliac region and, as recommended above, I incised in the right rectus, practically in the position of an incision for appendicitis.

The palpating finger readily found a mass in the lumbar-iliac region, which was with little difficulty delivered through the abdominal incision. This mass was found to consist of about two inches of the ileum, the cecum and the appendix being still intussuscepted, and to its proximal side near the ileocecal valve, a mesenteric lymph node about 1x½x½ inches, deeply congested, attracted immediate attention. Upon reducing the intussusception the appendix was noted as being thickened, intensely congested (almost black) and covered with some lymph flakes. Appendicectomy was deemed advisable and was done.

No sutures were made to the parietes of the gut or mesentery. The abdominal incision was closed in layers, with the final result as reported by Dr. Huber. The involvement of the lymph node was evidently only of recent date, due in a measure to the obstruction of circulation and possibly to the result of a mild infection from its contiguity to the involved area. This condition has been before observed by me in instances of strangulated hernia and simple appendicular cases.

In reducing the condition two things for success must be remembered.

First, never pull the proximal end with the idea that reduction will be accomplished. This type of manipulation is more productive of tears of the coats of the intestines than it is of producing the end desired. Second, and the more important point to remember, is that reduction is only produced by pressure upon the apex of the mass through the gut at the distal extremity, gently guiding the proximal end. Should adhesions between the entering and the receiving gut prevent the reduction, then the passing of a blunt instrument between these portions is usually attended by success.

Recovery can be assured in most cases of less than one day's standing; in cases of two days and more than one the majority recover; in cases of over two and up to three days a fair percentage recover, while in cases of over three days the prognosis is bad.

So far as I can recall from the literature to date, no case of

excision of intestines in these cases in children under one year has been successful. We may add that the mortality in all cases is from 20 to 50 per cent.

With these facts staring us in the face, the mortality rate can be greatly reduced if clinicians will look upon these cases as distinctly surgical in character. We should not temporize any longer but strongly advise and insist upon immediate surgical interference if injections carefully tried are not productive of immediate positive results in the early cases, that is, in those of less than twelve hours' duration.

Hydrostatic pressure or inflation by air should not be used in any case of over twelve hours' duration.

DISCUSSION.

DR. CAILLÉ.—For the clinician there are three main points of interest connected with this subject. First, the possibility of early diagnosis; second, the possibility of overcoming the obstruction by nonoperative means; and, third, the possibility of locating the site of the intussusception and thus aiding the surgeon. The diagnosis is easy in advanced cases, but very difficult when you see a case at the outset, and, I dare say that general rules cannot be laid down to guide us; each case must be judged on its merits. Dr. Huber is to be congratulated on his success in the early diagnosis of this condition, particularly as the patient was his own child and probably owes its life to his early recognition of a grave malady.

Regarding the second point, the possibility of overcoming the obstruction by nonoperative means, I would like to say that I have had most success in reducing intussusception by means of oil injections—the child being almost in an inverted position. I inject warm oil and gently manipulate the abdomen.

Regarding the third point, the possibility of locating the intussusception, that is of considerable importance because the surgeon is not going to open in the median line if he knows that the intussusception is located laterally. Some fifteen years ago I drew attention to the following percussion phenomenon in intussusception: If you percuss the abdomen in intussusception, before the tumor can be made out before the abdomen is highly tympanitic, you will generally find at the site of the constriction a *very tympanitic area* adjoining a dull area, and, in my experience this point, when found, will be the proper site for opening the abdomen.

DR. PACKARD.—One point in Dr. Huber's report was of spe-

cial interest to me. This was his mention of the presence of the large lymph node in the intussusception. Three or four years ago Dr. Le Conte operated for me upon a case of intussusception of six days' duration. It was found impossible on opening the abdomen to reduce the intussusception because of the existence of just such an enlarged lymph node close to the junction of the ileum and cecum. When we think of the proximity of the mesenteric lymph nodes to the intestine it is easy to see how even several of these might be drawn to the intussuscipiens and when swollen might seriously interfere with complete reduction.

DR. JACOBI.—Has any member of this society ever seen a perforation occurring after injection when the intestine was in a normal condition—when it was healthy? I should say no, for the reason that I have seen perforations in such cases only when, at least, the adventitia of the intestine was abnormal. In every one there was a previous history of diarrheas, sometimes constipation and diarrhea alternating, occurring months and sometimes years before the intussusception. There was thickening and discoloration of the adventitia corresponding with the perforations, and, therefore I think the question justified—whether the gentlemen have seen perforations in such cases where the intestines were perfectly normal?

In that connection it would be well to remember that in a good many of our cases of early enteritis, in its different forms, we have as a result a local, and sometimes a fairly general, peritonitis, that will show itself in after life in thickening and changes in the walls with tendency to perforation. In many cases of perforation from alleged unknown causes a local peritonitis will be found that has made the walls more friable.

DR. PUTNAM.—Dr. Jacobi has said that ruptures do not occur in a healthy intestine, but I should like to ask if rupture is not extremely rare even with any kind of an intestine? Can anyone mention such an accident? While these cases ought to be operated on, sometimes they cannot be operated on, and then, I think, pressure ought to be used to reduce them.

DR. GRIFFITH.—I would like to know how much importance Dr. Huber places upon the presence of intense pain in these cases; whether or not he considers it an essential. My own experience in some of these cases has been that one could be readily deceived by the absence of pain. I can recall at least two children who suffered no more pain than one would expect in the ordinary straining which frequently attends diarrhea of an inflammatory nature. There was no outcry at all. The diagnosis had to depend entirely upon the presence of the tumor and the bloody movements. Of course, the taking away of any one such important symptom increases the difficulty of recognizing the con-

dition, and yet I feel that we would make a mistake in claiming that intestinal obstruction must be attended by intense pain.

I wish to call attention to the existence of recurrent, or cyclic, vomiting as a condition which may readily be confounded with intestinal obstruction. Of course, where there have been previously repeated attacks of this nature, the diagnosis is easy, but with the first attack it is sometimes extremely difficult. I can recall one case in which the presence of intestinal obstruction was very greatly suspected by the physician in charge. The absolute constipation, the collapse, the repeated uncontrollable vomiting, so characteristic of recurrent vomiting, are equally well seen in obstruction.

DR. CHAPIN.—It is sometimes difficult to make a diagnosis between gastritis of a severe type and an intussusception in a baby. I reported a case some years ago that I had seen in consultation. A little girl, five years old, had intense vomiting that became stercoraceous. She had great pain and the pinched expression. No tumor was made out, but there was ballooning of the rectum. We washed out the bowel—she had at first been given laxatives which had not operated. The case looked so much like obstruction that I called in a surgeon who advised operation, which was declined. The patient died, and at the autopsy, we found an extreme condition of gastritis but no obstruction; there had been constipation because the child had nothing in the bowel. The whole picture looked to us like obstruction, and yet it was nothing but an acute gastritis.

DR. JACOBI.—Dr. Putnam says cases of perforation are very rare. I should say they are rare, but not unheard of.

In regard to the method of making injections I cannot accept the suggestion that increased pressure will accomplish more than gentle pressure. The intestine is not an iron tube and hydrostatic laws are not applicable. It is gentle dilatation of the intestinal wall around the part of the impacted mass that we want, and therefore irrigation with moderate pressure and manipulation under chloroform will accomplish very much more than increased pressure. Gentle dilatation is the best means of securing reduction. I remember that a physician in New York recommended that pressure should be made by having the column of water at a height of fourteen feet and that the bag should be taken up stairs to exactly fourteen feet, the height which he calculated arithmetically to be correct. Now I do not believe that is the correct thing for we have to deal with tissues that will dilate and the matter cannot be subjected to arithmetical calculation. I think the effect is better when the irrigation is slow and when the irrigator is kept at a height of only one or two feet so that the dilatation takes place very gradually.

DR. ADAMS.—I think a great deal depends, as has been said, upon making the diagnosis of a tumor. In the past eighteen months I have seen several cases of intussusception, all in children under nine months of age.

The first case was the infant of a physician who had treated the case during the night and who had practically made the diagnosis. The child was in a state of coma from opium and I was called for that more than for anything else. The narcosis was produced by a small dose of the deodorized tincture given by the rectum. Within a few hours I was able to make a diagnosis of intussusception which I located at the hepatic flexure of the colon. I insisted upon an operation, and about six inches of the colon was found intussuscepted. The child recovered and is now strong.

The second case was one in which the diagnosis had been made and I was called in consultation. I had no hesitancy in confirming the diagnosis, nor was there any difficulty in locating the tumor in about the same position—the hepatic flexure. Here there were no symptoms of obstruction. In the first case there were straining and crying, but in this one there were not, and in fact the child was crawling around on the floor when I went into the room. The family refused to have an operation and I left the case. Another physician was called who said there was obstruction but that he had been called too late or he could have stopped it without an operation. A surgeon was brought in the next day but he would not operate as the child was too far gone. A necropsy was permitted and it was found that about ten inches of intestine was intussuscepted and in a gangrenous condition. The surgeon said he doubted if operation would have been of any benefit even when I saw the child.

The last case was one occurring in a child three or four months of age, a perfectly healthy baby. It had an attack of intestinal trouble and was given a dose of castor oil by the mother. After that the child would strain, cry and break out in a profuse sweat, without the escape of mucus or anything else. I made a diagnosis of intussusception and tried the irrigation as spoken of by Dr. Jacobi—and that is the method I generally use, with the injection from a height of about one foot. I agree with him that you can get better results in that way. If you exert more pressure the contractile power of the gut is set up and you cannot force in as much water to produce dilatation. Now in this instance with one injection the gut was relieved, but two months afterward I was sent for to see the child in the same condition. I did not locate a tumor at the first attack, but in the later found the gut almost protruding from the anus and with my finger pushed it up and could feel it recede from the finger. I used a slow injection. The child recovered and has never had any return of the trouble.

My observation has been, so far as surgical procedures are concerned, that there are two classes of physicians—those who want to operate as soon as they see a case, and those who are more conservative and want to wait and see what the chances are. I think the practitioner is competent to make the diagnosis and when he makes it he is ready for operation if the family will consent to it, but we cannot always operate. The parents have something to say about it and often they will not permit an operation, so that we cannot always get the good results we would like when we know that an operation is the only thing. We must judge the case as it appears to us and I do not think the surgeon can lay down any rules that will guide us. We occasionally meet with a case in which none of the prominent symptoms are present.

DR. PUTNAM.—While I believe laparotomy to be the proper treatment for such cases, still, if injections are to be used at all, I cannot see how they can be of any great use without a plug to close up the anus, thus enabling one to make pressure. At another meeting I should like to show an elastic plug which is kept distended by the pressure of the water itself.

I should not be willing to think that there was nothing more to be done for a case when a small pressure of water would not relieve it and when I could not have an operation done. I saw a case many years ago when an operation would have been an alarming thing to propose. The child was nine months old and had been in that condition under the care of several physicians for two days. I tried for a great while to reduce the intussusception with injections from the ordinary height, but without the least effect and I should have had to go away and leave the child in that condition if I had not decided that it was better to run the risk of pressure. The bag was hung on the edge of the door, which makes about seven feet, and in a short time the tumor disappeared and the child was apparently cured. It was more comfortable and went to sleep, but in the morning the symptoms came on again and the tumor was again felt and I did the same thing over again. On the second occasion it took but a very short time with this pressure to reduce the tumor. So I should be very sorry not to have tried the higher irrigation. I think there is less danger than is generally supposed of rupturing the intestine because the abdominal walls act a good deal as the net bag around the thin rubber bulb of the atomizer, or cautery, which prevents a very thin bag from breaking whereas if that netting were not surrounding it the bag would become over-distended and burst.

DR. ADAMS.—You had not as much pressure as you would lead us to believe. You had not a height of seven feet because you had the child elevated and the bag hanging below the top of the door.

DR. CHRISTOPHER.—I should like to relate an observation that Dr. Henrotin made on one of my patients. The child was a boy four months old taken with the intussusception at nine o'clock in the morning. There was no pain, no tumor, but marked depression, and in the afternoon about two o'clock a little bloody discharge from the bowel. At six o'clock the abdomen was opened and the intussusception found. With the intussusception in sight we attempted to reduce it by introducing water into the bowel. We allowed the pressure to rise as high as we thought safe. It dilated the colon considerably without the least evidence of any reduction. Of course the conditions were not exactly the same as when the abdomen is closed, but we wanted to see, directly, the effect of pressure from within. It was completely negative. After this attempt the intussusception was reduced with the greatest ease by simply pulling the bowels apart. There were no adhesions. Three months later the child had intussusception again. This time the mother made the diagnosis and in nine hours we again opened the abdomen and reduced it, as readily as the first time, and with favorable result.

DR. BLACKADER.—Speaking as a physician, I think it would be a pity to have it go forth as the opinion of this Society that this condition may be treated by repeated injections, and that operation by the surgeon may be deferred. In my own opinion, the safety of the child depends upon prompt recognition of the intussusception and prompt operation. The cases in which injections succeed are very few and time is so important. I fully agree with what Dr. Huber says, that every hour counts, treatment of this condition by the introduction of either water or air into the bowels is a method upon which little reliance can be placed.

DR. ROTCH.—The Society has already discussed this subject in Cincinnati, and the opinion of the Society then was just what Dr. Blackader states, namely, that the operative treatment in intussusception is the best. It is merely a matter of chance whether an intussusception is reduced by irrigation or not. If you examine a large number of specimens in the museums, you will see a vast difference in the relations of the different parts of the intestines to each other. Where they are in line and you can bring direct pressure to bear upon them as you would on the inverted finger of a glove, the intussusception may be reduced in this way. Often, however, the parts are not in line and then the more pressure you exert with the fluid the greater you make the obstruction. It is absolutely contraindicated in such cases and it is mere chance whether the case is of this class or not. Exploratory laparotomies in children are now done in such a way that we do not hesitate in most cases to make use of this method for the purpose of diagnosis and there is no question that the best authorities all over the world are upholding the surgical

treatment of these cases. If you suspect intussusception it is perfectly harmless to use gentle dilatation, which will be of more service, in all probability, than greater pressure because with gentle dilatation you may reduce a kink in the intestine where with greater pressure you would make it worse. Of course, we have all had cases reduced by dilatation when seen in the early hours of the accident, before adhesions have formed. These are the successful cases. We should not wait, but as soon as we have tried the dilatation and have found that it is not successful, an exploratory laparotomy should be done at once. I think that this was formerly the opinion of the Society and I trust that it will continue to be.

DR. HUBER.—In addition to the lateral traction of the mesentery which disturbs the relation of the parts of intestine, spoken of by Dr. Rotch, there is another factor, and that is the rapid swelling of the intussuscepted portion. This proved to be the main difficulty in the case I report. Most of the cases we see are of the ileocecal variety, as statistics show. I can confirm the difficulty of differentiating between cyclical vomiting and early intussusception, unless you are familiar with the previous history of the case.

As regards the early symptoms they depend upon whether the case is acute, subacute, or chronic. My paper dealt with acute cases, which are the cases of greatest danger. The others give you time to make a diagnosis and there is not the same danger of strangulation, which is the chief element of mortality in the first class. When the case is acute the symptoms are due to strangulation; and pain, vomiting, change in expression and bloody or currant-jelly discharge are early symptoms.

As regards the presence of the lymph node, the enlargement was produced in part by interference with the circulation, and in part due to a mild toxemia induced by the intestinal lesion.

LOCAL VARIATIONS IN THE MORTALITY FROM SUMMER DIARRHEA.

BY HENRY DWIGHT CHAPIN, M. D.,

New York.

It is generally believed that the cause of the high infant mortality during the summer months lies in the milk supply, on the ground of the high bacterial content of the milk. In some respects this is so, but there must be some other factor or factors at work. In looking over the number of deaths from acute diarrheal diseases in New York State, to see if there was any connection between the temperature, or temperature and rainfall, and the number of deaths from diarrheal diseases, it was found that there was no apparent connection, for in some months with very high average temperature and low rainfall there were less deaths than in other months when the temperature was much lower and the rainfall greater. Seibert reached the same conclusion a number of years ago and stated that epidemics of summer diarrhea began when the minimum daily temperature reached 60°F. He accounted for this by reasoning that milk began to turn readily at this temperature.

New York State is divided into a number of districts by the State Board of Health. About one-half of the population of the State is in the maritime district which includes Greater New York and its suburbs. The number of deaths from acute diarrheal diseases were collected for this maritime district, and for the remaining portion of the State. In both portions of the State the number of deaths varies greatly from year to year, regardless of the temperature. It is impossible to suppose that throughout the whole State the methods of handling milk are better in the years with a small number of deaths, and poorer in the years with a large number of deaths.

Greater New York City draws its milk supply from a very wide area, which covers at least one-half of the entire State. If an infected milk supply were the only cause of the variation in

the number of deaths from diarrheal diseases, New York City should show it particularly, as its milk is much older than that used in the country districts. But it is repeatedly found that in years when there is a large number of deaths in the country districts there is a small number in the maritime district, and in other years when there is a great number of deaths in the maritime district the number of deaths in the country district is very small. For instance, the number of deaths from acute diarrhea in the country and maritime districts was for the months of May to October inclusive:

YEAR.	COUNTRY DISTRICT.	MARITIME DISTRICT.
1899	2,187	3,557
1900	3,202	3,867
1901	1,898	6,115

In the country there was in 1900 an increase of over 50 per cent., and in the maritime district of less than 10 per cent. over the deaths of 1899. In the country in 1901 there was a decrease of 40 per cent., and in the maritime district an increase of 58 per cent. in deaths compared with 1900. In 1896 the country deaths increased 10 per cent., while the deaths in the maritime district decreased 10 per cent. compared with the figures for 1895.

At all seasons of the year there are large numbers of bacteria in milk, so mere numbers can play but a small part.

The most common bacterial change in milk is souring; sour milk is not necessarily an unwholesome article of food for adults, but milk that is just beginning to sour forms exceedingly tough curds in the stomach under the action of rennet, which on this account renders it unfit for infant feeding.

During the months of July and August milk is particularly liable to undergo a gassy fermentation, which causes a rennet curd of milk to become spongy and appear very much like a mass of unbaked bread dough. As far as is known, the souring and gassy fermentations of milk are caused by bacterial action on the sugar of the milk. Poisonous products are not thus produced, but these fermentations may and probably do cause indigestion in infants.

There is another type of fermentation in milk that is less common, in which the proteids of the milk are attacked, foul smelling gases and poisonous compounds being formed. Most

DEATHS FROM ACUTE DIARRHEAS

MARITIME DISTRICT

NEW YORK, KINGS, QUEENS, RICHMOND, WESTCHESTER COUNTIES

MONTHS	DEATHS	MEAN TEMPERATURE	HIGHEST TEMPERATURE	LOWEST TEMPERATURE	GREATEST DAILY RANGE	RAIN FALL INCHES	HUMIDITY
1892. MAY...	82	60.0°	81°	42°	31°	4.30	74
" JUNE...	588	72.0	91	56	25	2.90	—
" JULY...	2961	74.8	96	56	23	2.45	70
" AUG...	1339	73.9	93	59	21	3.90	74
" SEPT...	670	66.0	83	51	25	0.87	69
" OCT...	303	55.4	79	39	26	0.63	66
Total.....	5943						
1893. MAY...	120	59.0°				5.06	
" JUNE...	397	69.0				2.56	
" JULY...	2504	75.0				1.26	
" AUG...	1334	72.0	93°	58°	24°	7.02	72
" SEPT...	755	64.0	84	45	25	2.27	72
" OCT...	362	57.6	78	34	28	5.28	75
Total.....	5472						
1894. MAY...	90	60.8°	83°	44°	28°	3.90	74
" JUNE...	652	70.6	94	48	25	0.86	70
" JULY...	2403	76.1	96	58	25	2.89	69
" AUG...	1065	73.0	90	55	26	1.54	70
" SEPT...	706	69 6	94	46	22	8.04	78
" OCT...	328	57.2	75	30	23	5.83	73
Total.....	5244						
1895. MAY...	103	59.4°	95°	38°	30°	2.04	73
" JUNE...	512	70.0	95	52	33	2.57	79
" JULY...	2258	71.0	89	55	21	4.40	72
" AUG...	1405	74.0	90	55	21	4.12	—
" SEPT...	983	69.7	97	44	23	0.95	72
" OCT...	398	51.0	71	34	21	4.04	70
Total.....	5559						
1896. MAY...	145	64°	91°	43°	38°	2.01	74
" JUNE...	729	66	87	51	22	6.38	80
" JULY...	1990	73	89	59	23	4.45	82
" AUG...	1314	73	94	56	23	2.46	78
" SEPT...	525	65	86	43	24	3.04	80
" OCT...	205	52	72	36	25	1.71	79
Total.....	4908						

DEATHS FROM ACUTE DIARRHEAS

MARITIME DISTRICT

NEW YORK, KINGS, QUEENS, RICHMOND, WESTCHESTER COUNTIES

MONTHS	DEATHS	MEAN TEMPERATURE	HIGHEST TEMPERATURE	LOWEST TEMPERATURE	GREATEST DAILY RANGE	RAIN FALL INCHES	HUMIDITY
1897. MAY. . .	61	59°	79°	44°	23°	5.30	70
" JUNE. . .	425	65	86	48	21	2.98	68
" JULY. . .	1938	73	89	60	21	9.52	82
" AUG. . .	1010	71	84	60	18	3.14	76
" SEPT. .	654	65	91	44	26	1.64	72
" OCT. . .	252	56	86	37	27	0.72	73
Total.	4340						
1898. MAY. . .	80	56.5°	86°	38°	22°	5.55	80
" JUNE. . .	285	68.9	89	53	24	1.28	69
" JULY. . .	1738	74.0	99	57	29	4.76	77
" AUG. . .	1481	74.3	90	60	17	3.12	78
" SEPT. . .	979	68 8	94	49	22	1.28	76
" OCT. . .	305	57.6	80	38	25	6.14	79
Total.	4868						
1899. MAY. . .	69	61 °	84°	46°	27°	1.14	
" JUNE. . .	555	72.2	97	55	31	1.83	
" JULY. . .	1491	73.8	90	59	22	6.20	
" AUG. . .	809	73.6	91	60	21	3.90	
" SEPT. . .	472	65.2	84	46	20	5.89	
" OCT. . .	161	58 2	79	36	22	2.05	
Total.	3557						
1900. MAY. . .	86	60.8°	89°	38°	31°	4.05	62
" JUNE. . .	371	71.4	91	56	22	3.36	70
" JULY. . .	1459	76.4	94	58	27	4.33	72
" AUG. . .	967	76.8	95	60	28	2.69	71
" SEPT. . .	691	70.8	90	53	18	2.36	72
" OCT. . .	293	60.8	77	37	29	4.17	79
Total.	3867						
1901. MAY. . .	188	58.6°	82°	44°	27°	7 01	74
" JUNE. . .	371	71.3	97	53	25	0 94	66
" JULY. . .	1831	78.1	99	64	24	5.41	75
" AUG. . .	1891	75.6	88	63	20	6.88	77
" SEPT. . .	1314	68.4	87	49	23	2.33	74
" OCT. . .	520	56	75	38	24	2.20	69
Total.	6115						

DEATHS FROM ACUTE DIARRHEAS

NEW YORK STATE *EXCEPT* NEW YORK, BROOKLYN, LONG ISLAND, STATEN ISLAND, WESTCHESTER COUNTIES

(MARITIME DISTRICT)

MONTHS	1892.			1893.			1894.			1895.			1896.		
	DEATHS	MEAN TEMP.	RAIN FALL INCHES	DEATHS	MEAN TEMP.	RAIN FALL INCHES	DEATHS	MEAN TEMP.	RAIN FALL INCHES	DEATHS	MEAN TEMP.	RAIN FALL INCHES	DEATHS	MEAN TEMP.	RAIN FALL INCHES
MAY	31	54.2°	5.60	55	55.°	5.74	46	56.8°	5.·0	54	58.7°	2.60	60	61.0°	2.52
JUNE	88	65 0	2.54	81	67.7	6.46	87	66.8	3.41	114	69.3	2.75	186	64.7	3.19
JULY	668	69.2	4.56	702	68.9	3.29	855	70.9	2.58	716	66.8	3.12	1096	70.4	4.90
AUGUST	989	68.1	6.48	1072	68.	6.32	1003	65.7	1.61	898	68.1	4.11	1012	68.9	2.98
SEPTEMBER	586	59.6	2.35	640	57.0	3.20	748	63.8	4.97	689	65.0	2.31	552	59.4	4.63
OCTOBER	188	48.2	1.71	171	51.0	2.41	307	51.2	4.63	256	44.0	1.94	133	46.1	2.57
Total	2550			2721			3046			2727			3039		

MONTHS	1897.			1898.			1899.			1900.			1901.		
MAY	52	55.6°	4.14	47	56.1°	4.45	53	57.2°	2.90	71	55.8°	2.35	48	55.6°	5.03
JUNE	78	61.6	3.63	87	66.8	2.96	131	67.5	2.39	106	66.6	2.63	79	66.7	3.35
JULY	458	72.1	6.81	560	72.2	3.17	577	70.3	3.72	623	70.6	4.10	400	73.0	4.28
AUGUST	789	66.0	3.06	864	70.0	6.14	726	69.3	1.88	1018	71.4	3.24	664	69.0	5.11
SEPTEMBER	517	60.3	2.01	893	63.8	3.10	500	59.2	4.46	965	64.0	2.27	494	61.4	3.46
OCTOBER	192	51.0	1.06	382	51.5	5.19	200	52.5	2.27	419	55.9	3.05	213	49.7	2.03
Total	2086			2833			2187			3202			1898		

of the bacteria that attack proteids, the putrefactive varieties, are spore bearing and consequently liable to be carried in dust while the souring and gassy varieties are not thought to be spore bearing, and hence not so likely to be carried in this manner.

It is certain that the great increase in the number of deaths in New York City in the summer of 1901, cannot be attributed entirely to the milk supply, or to the heat, for the heavy mortality was in August, September and part of October, and not during the excessively hot weather of the latter part of June and early July. In and around New York City there was in the summer of 1901 the greatest number of deaths for ten years, and during the same period the least number of deaths in the country. During the previous summer there was next to the lowest number of deaths for ten years in the city district, and the greatest number for ten years in the country. If the primary source of infection was the milk supply the deaths in the city districts should have shown at least a proportionate increase in the summer of 1900, and the country districts should have had a large number of deaths in 1901.

It is conceded by all that the intense poisoning cases of summer diarrheas are the result of putrefactive changes in the intestinal contents, excessively foul stools accompanying high fever as a rule. The great source of putrefactive bacteria is the *soil* which consists of a mixture of decomposing organic matter and earth. Dust contains great numbers of putrefactive bacteria and spores, and it is from such dust and dirt that milk becomes infected with putrefactive bacteria. Theoretically we should expect to find a great deal of summer diarrhea in localities where soil rich in decomposing matter is being turned over and dried so that it can be carried by the wind as dust or possibly by flies. This may account for the occasional infection of breast-fed infants.

With the removal of horse cars and with cleaner streets in Greater New York the number of deaths from diarrheal diseases had on the whole been steadily declining for the past ten years, and at the same time the population had been increasing. In 1901, the Borough of Manhattan, was torn up from one end to the other for the purpose of building the subway; water pipes and sewers were opened and changed, and the streets were very dusty.

It is certainly a remarkable coincidence that with an unusually small number of deaths from diarrheal diseases in the remaining portion of the State, the steadily declining number of deaths from the same cause in the city district should suddenly increase 58 per cent., when the streets were torn up.

There can be no doubt that in preventing diarrheal diseases in summer, attention must be paid to local conditions as well as to improving the milk supplies.

DISCUSSION.

DR. SEIBERT.—The author of the paper has divided the State of New York into country and maritime districts. The only valuable statistics we can get in regard to the milk supply is by taking a city where the mode of supply is carried on in a uniform manner. The State of New York is so extensive, a whole country in itself, and the conditions for the milk supply as well as of the weather vary so much that I do not think this classification can be of value.

As to the matter of temperature and rain-fall, the author's statistics, I believe, are antiquated. I have shown in my work on cholera infantum and the weather (*Medical Record,* March, 1888) that it is misleading to compare mean monthly temperatures with mortality or morbidity statistics. In 1878 (for instance) in the month of August occurred the greatest rain-fall in New York for many years—7.2 inches in three days; the rest of the days were clear and bright and not a drop of rain fell. Was that a wet or a dry month? My investigations were carried on so that not alone the temperature and other weather constituents of each day of ten years were taken, but also three observations of each day, and charts were made showing the conditions. I think this question was settled by me, for European investigators at least, that monthly sums and averages cannot be used in scientific work of this kind any more. Dr. Chapin said that in a certain year 1,800 children died in August and in July 1,831. In my work I had found a law which holds good for the principal cities of Europe and America, that practically in each July more children die of summer complaint than during the following August. There may be exceptions as in this one year, as the author has pointed out. From 1878 to 1888 about 1,200 children died in New York during every July of summer complaint and during the following August usually not more than half that number, no matter whether the temperature was higher or not. This law has a simple explanation: if 1,200 babies die during July in one city, then in the following August there are 1,200 babies less to get sick.

Dr. Chapin says this abnormal mortality of last summer may bear some relation to the digging up of the streets in New York in making the underground railway. Possibly so, but if you remember that the milk is not carried around in open but in closed cans through the streets, then I hardly think we can make that connection. The main source of infection of milk is to be found during milking, when cow's manure falls into the open pail.

DR. ADAMS.—There are many factors responsible for infant mortality in summer. The meteorological conditions play their part and I think if the humidity is studied with the temperature it will be found that it does hold some relation to the cause. Dr. Busey of Washington, D. C., published a work in which the meteorological conditions in relation to infant mortality in the cities of New York, Philadelphia, Washington and New Orleans, were studied and his conclusions were published in that work. I was associated with him at that time and had an opportunity to study this subject. Now so far as heat as a factor is concerned my observation is that during the prolonged hot spells in which the temperature averages 96° for the day, that the children are not sick during that time, but it is when the humidity is great and when the temperature falls. You cannot slight any one of these conditions.

DR. COTTON.—A month is an arbitrary thing when you compare one month with another and say the mortality of this month is so much and the temperature and humidity so much. I do not think statistics of this kind are satisfactory. Suppose the temperature falls on the 30th day of July and the patient dies on the 5th day of August, is the death due to the conditions pertaining in August or to the conditions in July? What right have we to assume that deaths in July are due to conditions in that month? They may have been due to conditions in June.

DR. CHAPIN.—I have only tried to show the causes operating here in New York and have not tried to lay down rules for St. Petersburg and London. I have said what caused the variation. In Greater New York in 1901, 6,115 cases died from summer diarrhea—in 1900, 3,867. These tables can be studied and the members draw their own conclusions.

A CASE OF CHONDRODYSTROPHY FETALIS.

BY JOHN LOVETT MORSE, A.M., M.D.,

Instructor in Diseases of Children, Harvard Medical School; Assistant Visiting Physician at the City Hospital and at the Infants' Hospital, Boston.

John N. was brought to the Boston City Hospital when two months old and was turned over to me by the courtesy of Dr. F. B. Lund.

FAMILY HISTORY.—His parents were Italians, well, and of fairly good habits. There had been no history of such deformities in either family. There had been no miscarriages. The first child weighed twelve pounds, and was born dead after a severe instrumental delivery. The mother gave a history of seeing a dwarf while carrying this, the second child.

PERSONAL HISTORY.—He was born at full term, feet first, before the arrival of the doctor. Except for his deformities he seemed normal. He was given only the breast and, although he had not gained rapidly, he had had no marked disturbances of the gastroenteric tract. He cried a good deal and was brought to the hospital because of an inguinal hernia.

PHYSICAL EXAMINATION.—He was small, weighing only six pounds, two ounces. The flesh was fairly firm and the skin and subcutaneous tissues normal in character. He seemed as intelligent as the average baby of his age.

The most striking thing about him was the comparative shortness of the extremities, especially of the legs. (See Fig. I.)

His measurements were as follows:

Length—Vertex to sole	42	cm.
Vertex to navel	25	"
Vertex to anterior superior spine	28½	"
Anterior superior spine to sole	13½	"
Head—Diameter—Glabella-occiput	10	"
Bitemporal	9	"
Biparietal	11	"
Circumference—Glabella-occiput	33	"
Occipito-frontal	33½	"
Neck—Circumference	18½	"

Chest—Circumference at nipples	30½	"
Abdomen—Circumference at navel	30½	"
Pelvis at crests	26	"
Extremities—Arm—Acromion to external condyle	7	"
Ulna	7	"
Leg—Trochanter to external tuberosity (lowest portion)	6½	"

The bridge of the nose was depressed, but possibly not beyond normal limits. The frontal suture was open about half way down the forehead. The coronal suture was open nearly to the level of the eyebrows. The frontal bones were of normal density. The parietal bones were prominent and of normal density. Their posterior borders were thicker than normal. The back of the head was flattened. No bony structure could be felt in the occipital bone above the protuberance. The eyes were normal except for slight exophthalmos. There were no snuffles. The tongue was small. The neck not unusually short. The ears were normal. Nothing abnormal was detected about the thyroid. The clavicles were small. There was very slight beading of the ribs. The scapulæ were normal. There was nothing abnormal about the spine. The heart and lungs were normal. There was nothing noticeable about the abdomen except a slight umbilical hernia and a large left inguinal hernia. The liver was normal. The spleen was not palpable. The feet and hands were normal in shape but unusually small. The extremities were short, especially in the thighs and upper arms. The X-ray photographs show the condition of the bones better than any description. (See Figs. II., III., IV.)

The humerus felt at least 2½ cm. thick. The rotation of the radii was normal. All motions at the wrists were normal, as were those of the hands. There seemed, however, to be a tendency to keep the hands flexed at the wrists with the fingers extended.

Flexion at the hips was normal, but extension was somewhat limited. He lay with the thighs rotated outward and somewhat flexed. The trochanters were easily made out. The patellæ were very small. Extension at the knees was a good deal limited and the knees were held somewhat flexed. The thighs and legs were held almost in the position of a circle. It was impossible to distinguish the separate bones in the legs. The motions at the ankles and of the feet were normal.

There was a slight, general enlargement of the lymph nodes.

He died of some enteric disease when four months old. Unfortunately an autopsy was not obtained.

FIG. I.—PHOTOGRAPH SHOWING THE SHORT EXTREMITIES.

CLASSIFICATION.—The class of cases to which this belongs has been described under many names, among which may be men-

FIG. II.—X-RAY PHOTOGRAPH OF THE LEFT ARM.

tioned fetal rickets, osteogenesis imperfecta, achondroplasia and chondrodystrophy fetalis. In a general way, in these cases, the trunk is normal, the extremities short and deformed, the head large and the bridge of the nose flattened. Although a considerable number of cases has been thoroughly worked up, both

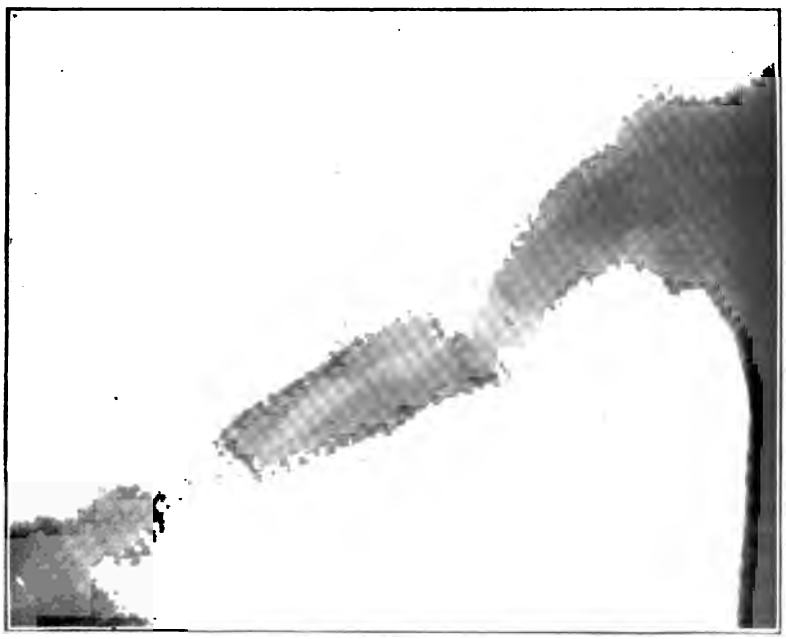

FIG. III.—X-RAY PHOTOGRAPH OF THE RIGHT ARM.

macroscopically and microscopically, by competent observers, no scientific classification on pathological lines is at present possible. While the pathological lesions in the different specimens resemble each other in a general way, no two cases show exactly the same changes. Certain points, however, seem pretty well established.

FIG. IV.—X-RAY PHOTOGRAPH OF THE LOWER EXTREMITIES.

PATHOLOGY.—The most important pathological process is, in brief, a disturbance of the normal process of ossification of the primary cartilage. It takes place early in fetal life, probably most often between the third and sixth months, and has ceased before birth. Hence, chondrodystrophy fetalis seems the most suitable and comprehensive name to apply to it. Kaufmann has described three main types of fetal chondrodystrophy: Chondrodystrophy

hypoplastica, chondrodystrophy malacica and chondrodystrophy hyperplastica. In chondrodystrophy hypoplastica the principal trouble is a simple failure of development of the cartilage; in chondrodystrophy malacica there is an abnormal softening of the cartilage and in chondrodystrophy hyperplastica there is an extremely irregular and immoderate development of the cartilage in all directions. Osteoporosis or osteosclerosis may occur as complications in any case. Fractures may also increase the deformities.

As the pathological process usually affects endochondral ossification only and is active only during the earlier months of fetal life, only those bones are involved in which this method of ossification normally takes place at this time. In them, however, the process is most definite and universal. Those bones which are formed entirely in membrane and those which, though formed in cartilage, remain entirely, or almost entirely, cartilaginous until the latter part of intrauterine life, are almost never involved. The periosteal formation of bone goes on, moreover, normally or more actively than usual. These facts explain the distribution and, to a certain extent, the character of the deformities seen in these cases. It is for these reasons that the bones of the upper part of the cranium, of the trunk (except the ribs) and of the carpus and tarsus are normal, while those of the base of the skull and of the pelvis as well as the ribs and the long bones of the extremities are deformed. Certain peculiarities seem worthy of more detailed consideration.

Head.—The flattening of the bridge of the nose is due to the shortening of the base of the skull. This shortening may be due either to premature tribasilar synostosis or to imperfect development of the basal cartilages. The increase in the size of the cranium is partly apparent and partly real; apparent because of the small base, and real because of the larger amount of room required in the cranium by the brain on account of the small base.

Thorax.—The flattening of the sides of the chest seen in some cases is due to the arrested development of the ribs. The costal cartilages are normal. The beading of the ribs is due either to periosteal overgrowth at the end of the diaphysis forming a cup-shaped mass around the epiphysis or to the displacement forward of either cartilage or rib by an asymmetrical development resulting from the abnormal folding-in of the periosteum.

Pelvis.—The pelvis is, as a rule, contracted and flattened. This is due to the abnormal ossification of the innominate bones. The growth of the sacrum and coccyx is not interrupted.

Extremities.—The long bones of the extremities depend upon cartilaginous ossification at the junction of the epiphyses and diaphysis for their growth in length during intra- as well as extra-uterine life. Any arrest of this process results, therefore, in shortening of the extremities. In chondrodystrophy the chief pathological changes are at the line of ossification. The most important of these are in the columnar zone, which is absent or defective. Growth in length is thus prevented. There is, in addition, in some cases, a fibrous ingrowth of the periosteum between the epiphyseal cartilage and the diaphysis. This prevents, of course, any further growth in length at this point.

The shafts of the long bones are almost entirely composed of periosteal bone. This is often excessive and not infrequently abnormally hard. The medullary canal is sometimes diminished in size or even replaced by hard bone. At the ends of the shaft the periosteal overgrowth may extend peripherally so as to form a cup about the cartilaginous extremity. This periosteal overgrowth is often mistaken during life for rachitic overgrowth.

Bowing of the long bones is common. The pull of the muscles accounts for this when the bones are softened. When they are hard the bowing is probably due to local intrusions of the periosteum which prevent regular growth, and to a resistance of the thick cartilages at the epiphyses to the growth of the diaphysis.

DIAGNOSIS.—It is evident that there is nothing in common between chondrodystrophy and rickets except the macroscopical appearances. In them there is a marked resemblance. Microscopical examination, however, shows that the pathological changes which produce such similar appearances are radically different. For example, the enlargement of the ends of the long bones in rickets is due to the abnormal development of the epiphyseal cartilage; in chondrodystrophy it is due to periosteal overgrowth. Microscopically, in rickets, the proliferating zone is much wider than normal; in chondrodystrophy it is narrower than normal. In rachitis vascularization is marked; in chondrodystrophy it is not marked. The changes in the skull, as already noted, are different in the two diseases. In rachitis the bones laid down in membrane as well as those in cartilage are involved; in

chondrodystrophy they are not. The term "fetal rickets" should not, therefore, be applied to this condition. It is possible, however, that true rickets may occur in intrauterine life, recovery taking place before birth. Such a process would deserve the name of "fetal rickets." Its occurrence seems hardly probable, however, as the time is very limited for the development and "cure" of rickets, even if of a mild type.

While there must be some doubt as to the existence of true "fetal rickets," "congenital rickets," that is, rickets beginning during intrauterine life and continuing into extrauterine life, undoubtedly occurs. The only question is as to its frequency. Kassowitz found evidences of rachitis in more than one-half of new-born infants, while Feyerabend found them in 69 per cent. of 108 cases. Unruh, moreover, claims that rachitis is always a congenital affection. Fédé and Finizo, on the contrary, of 475 new-born infants, found only three which showed clinical characteristics of rickets. Three others showed craniotabes. Fifty-two per cent. showed irregularities in the bones and sutures of the skull. They examined microscopically the bones of the skulls of six babies dead born or dead soon after birth. In none of them did they find rachitic lesions. They conclude that in the new-born, large fontanelles and wide sutures are not always indicative of rickets, and that in the majority of cases, at least, they simply indicate retarded ossification. Tschistowitsch examined the skeletons of 100 infants dead at, or shortly after, birth. In no case did he find evidences of pure rickets. In thirteen there was some doubt as to whether there was syphilis or rickets or a combination of both. Fifteen cases showed syphilitic osteochondritis. He concludes that, histologically, rickets and syphilis have been often confused.

It has been assumed on account of the shortened base of the skull that chondrodystrophy and cretinism must be intimately related; in fact, some authors have gone so far as to say that chondrodystrophy is a manifestation of cretinism. Too much stress has been laid on the shortened base of the skull, however, as it has been assumed that it must be due to basilar synostosis, and that that is pathognomonic of cretinism. Shortening of the base of the skull may, however, be due to failure of development as well as to synostosis. This is usually the case in chondrodystrophy and may also be the case in cretinism. The pathological

changes in the long bones are dissimilar in the two diseases, and in chondrodystrophy other signs of cretinism are lacking. Stoeltzner, however, has reported a case in which the thyroid and tongue were enlarged and the subcutaneous fat tissues were hypertrophied. The extremities were short and microscopically showed dystrophy of cartilage. In this case, however, the dystrophy of cartilage seems better explained by a combination of the two conditions than as a symptom of cretinism.

Other evidence against any relationship between chondrodystrophy and cretinism is as follows: In no case have the parents of a case of chondrodystrophy been cretinistic. No case has as yet been reported in regions where cretinism occurs. Cases with chondrodystrophy have, however, borne children with the same disease. In cretinism the intelligence is subnormal; in chondrodystrophy it is normal.

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ETIOLOGY.—Little is known as to the cause of this disease. It has been attributed to privation, hardship and injuries during pregnancy, alcoholism, syphilis, cretinism, consanguinity and heredity. Privation and hardship during pregnancy have been noted in a few cases, but in others the conditions have been good. Moreover, privation and hardship are common and this disease is very rare. The same criticism applies to alcoholism and syphilis as etiological factors. Syphilis, moreover, causes pathological lesions in the bones of a different nature. Injuries could hardly cause such symmetrical and multiple lesions even if microscopical examination did not show that the pathological changes were not due to mechanical causes. Pregnancy in two of the cases (Kaufmann and Lampe) was the result of incest. The assumption of consanguinity as a cause is hardly justifiable on this basis, however.

In a considerable number of cases adults afflicted with this condition have borne children of the same kind. (Vrolik, Porak and Lauro.) In other cases, however, their children have been normal. (Charpentier, Boeckh, Porak, Friedenheim.) In the majority of cases, however, the condition did not exist in the parents, and even when these cases have borne children similarly deformed their own parents were normal. Similar conditions occur in animals and in some instances the deformities have been reproduced for many generations. While, therefore, the influence of heredity cannot be denied, it does not seem an important one and certainly does not furnish a satisfactory explanation as to

the essential cause of the disease. In fact, nothing is really known as to the essential cause. As in most of the cases the pathological changes have evidently taken place at the time of the ossification of the primitive cartilages and have come to a stop before birth, the disease must be active between the third and sixth months. As it is often associated with other defects of development, it must be due to some general cause affecting the nutrition of the organism as a whole. The association with hydramnios in some cases suggests that the disease may sometimes be due to alterations in the placenta. It is more probable, however, that both are due to the same cause, that is, some morbid process attacking the ovum as a whole. It is probable that this process may be of an infectious nature. Ballantyne suggests that the same cause acting early enough in intrauterine life may produce teratologic monstrosities, while if it acts later it may cause chondrodystrophy.

PROGNOSIS.—Chondrodystrophy affects not only the osseous system but also the general nutrition of the fetus. It is also often associated with other malformations. Hence in most cases the fetus dies in utero.

On account of the malformations, prolapse of the cord, faulty presentations and death during delivery are not infrequent. Even if born alive the child has, as a rule, as the result of the general disturbance of nutrition, but little vitality. The great majority die within a few days after birth.

The milder cases, however, may reach adult life. Edgeworth reports a man of fifty-nine; Thompson, men of thirty-nine and thirty-six; Porak, a man of twenty-seven; Crimail, a man of twenty-two, and Charpentier, one of thirty. The general characteristics presented at birth are retained to adult life. The extremities remain short, but the trunk develops normally. The heads are often unusually large. Muscular development is normal or greater than normal. The intellectual development is normal. The reproductive functions are developed at the normal age and are normal both in males and females. (Cases of Edgeworth, Thompson, Porak, Charpentier and several others.) In women, on account of the imperfect development of the pelvis as the result of this disease, childbearing is, however, a source of much danger. A number have had normal deliveries. In most, however, delivery has been instrumental or by Cesarean section. Some have borne

normal children (Charpentier, Porak, Friedenheim), others have given birth to children showing the same deformities (Lauro and Vrolik). In 1 case a female dwarf whose family were dwarfs with similar proportions gave birth to a normal child (Boeckh). In another case (Franqué) a woman had one normal child, one with club feet and three with chondrodystrophy.

AMERICAN CASES.—Very few cases of this condition have been reported in this country and the diagnosis of several of these is open to considerable doubt. They are as follows:

The first case reported in this country was by Jacobi. The case ordinarily referred to (*American Medical Times,* New York, 1863, VI., 127) is not a case of this disease. Dr. Jacobi writes that he has reported such a case but cannot remember whether it was published or not. I have been unable to find it.

Hirst, in 1894, under the head of "Rachitis Congenita Micromelica," gives the photograph of a specimen in the Wistar and Horner Medical Museum of the University of Pennsylvania. The history of this case is lacking. It is hydrocephalic. The limbs are short, curved and thick but nowhere fractured. The pelvis is rachitic in shape.

Smith reports a case of Dr. Heitzmann. The mother inhaled the vapor of lactic acid in her daily occupation during her pregnancy. The infant was born at term but died at once. The skull bones were completely absent and in the cartilages of the bones of the extremities and in those of the ribs there were scanty depositions of lime salts and numerous infarctions. (This case seems doubtful.)

Mason's case was a female, which lived three days. The mother's condition during the pregnancy was good, and there was no history of syphilis or rickets. There was almost no ossification of the parietal bones, the upper portion of the head containing only a few Wormian bones. The circumference of the head was twelve and a half inches. The frontal bones were partly developed. The temporal, occipital and facial were normal. The clavicles were small. There was a fracture in the left. The epiphyses of the ribs were enlarged. Both humeri were curved, the convexity being inward. There was a fracture in the left. The pelvis was undersized. The femora were curved, the convexity being outward. The ends of all the long bones were enlarged, the middle portions being deficient. The tibiæ were sharply curved with the

convexity forward. The hands were well developed. There was tongue-tie and exophthalmos. The color was dusky. There was no autopsy.

Townsend's case was a male which weighed seven pounds. The length was seventeen inches. It was born a month premature and died on the ninth day. There was no syphilitic nor rachitic history. The mother was not properly nourished during the pregnancy. The head measured thirteen and a quarter inches. The sutures were wide open. There was but little ossification of the cranial bones and less of the occipital, which showed marked craniotabes. The lungs were atelectatic, the sides of the chest depressed. There was marked enlargement of the epiphyses of the ribs. The humeri and the bones of the forearms were curved anteriorly. The femora were curved outward and forward. There were angular curvatures of the lower legs and a double inguinal hernia. There were complete fractures of both tibiæ, of the left humerus and of both bones of the right forearm. All united quickly. There was no autopsy.

Safford reports a case first seen when four and one-half years old. It was said to have been "all out of shape" at birth. The parents were Italians, and well. It was born at full term.

Smith reports a case of an infant born at term which died a few hours after birth from atelectasis. There was no syphilitic history. The skeleton is in the Wood Museum of Bellevue Hospital.

Smith reports another case, the child of a healthy mother, which died in five hours. He gives photographs of sections of the bones. Microscopical examination by Prof. Prudden showed no rachitic changes.

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DISCUSSION.

DR. JACOBI.—I would like to ask Dr. Morse if the description of the body given on the last page of his paper refers to rickets, or to chondrodystrophy?

DR. MORSE.—Chondrodystrophy.

DR. JACOBI.—If I were not told that I should have said it was a case of rickets, as it agrees with what you would expect in that disease, the shortening of the limbs, thickening of the ends of the bones, etc., exactly what you would expect in a characteristic case of rickets that has gotten well. Altogether the points in the paper that are meant to be differential between the two diseases are so numerous and were gone over so rapidly that I find it impossible to remember them all, but there were some points that certainly would not exclude a diagnosis of rickets. For instance, I think the reader of the paper said that in chondrodystrophy the swelling was periosteal, which is certainly also so in rickets. Therefore, is it the doctor's opinion that when the swelling is periosteal the disease is chondrodystrophy and not rickets? I should say that that could not be a diagnostic point. It is characteristic that rachitic bones remain thicker and shorter. The shortening is due to early ossification of the epiphyseal line where the growth of bone has its origin, and I think we have a right to claim this as rachitic unless there are other reasons why we should not do so. We should be very careful in differentiating such cases unless they are very, very marked. It appears to me that this case could well pass for rickets and nothing else. Another point is the retraction of the root of the nose and shortening of the base of the cranium. That could be, and is mostly, due to the early ossification of the synchondrosis between the occipital and sphenoid bones, thus interfering with the growth of the base of the cranium. It is the final stage of rachitis which ran its full course before birth. Cretinism used to be explained in that way by this premature synostosis, at a time some fifty years ago (for instance by Baillarger), when we did not know anything about the thyroid and the studies in regard to goiter were of an exclusively anatomical nature. It was looked upon as being due to the same cause as this form of rickets, developed before birth, and where the undue hardening of the bone took place before, or immediately after birth. It appears to me that we should be very cautious about the diagnosis of chondrodystrophy. There are undoubtedly such cases, but it has not appeared to me that the many points given us to-day as contributing to a differential diagnosis are very conclusive. It is very difficult to establish a differentiation. This much is plain, that in chondrodystrophy the epiphysis is large and the epiphyseal line thin and ossified at an early date. In these cases a large amount of fat is formed about the chest and about the limbs, the labia, eyelids, cheeks

and lips; the nose is flat and thick, the tongue large and deformed, exactly such as you see in infantile myxedema. Such a fetus looks exactly like a myxedematous child. I appreciate the paper and the care with which it has been drawn up, but it appears to me that I should be very willing to claim this case as one of rickets and not of chondrodystrophy.

DR. GRIFFITH.—The past year I have seen 2 cases which can reasonably be placed in the class described by Dr. Morse. One came to the Clinic of the University of Pennsylvania. Unfortunately, it died before we succeeded in getting photographs. The second I saw within a week at the Children's Hospital. Of this I was fortunate in getting photographs, but have not yet had time to have them printed. I was struck by the similarity between the feet in this last case and those in the pictures of Dr. Morse. There is the same pudgy, mole-like foot with a deep line at the spot where the instep joins the ankle. In my case there seemed just here to be a sudden thinning of the cutaneous and subcutaneous tissue. The hands were also very short, suggesting strongly the appearance of the mole's foot or of a paddle. The arms and legs were also short, though not as markedly so as in many reported cases. There were no signs of rickets at all, as far as I could discover.

There are several diseases of the bones seen in early life which, although, I believe, entirely distinct, yet touch each other in certain respects. Among these I may mention fetal rickets, achondroplasia, osteogenesis imperfecta and idiopathic osteopsathyrosis or fragilitas ossium. First as to fetal rickets; if there really is such a thing, it certainly appears to be rare. Fédé and Finizo carefully studied the heads of many new-born infants, making measurements of the fontanelle, and came to the conclusion that fetal rickets must certainly be rare and Tschistowitsch, as a result of a microscopical study of the bones of one hundred infants, likewise considers it uncommon. It probably differs in no respects from rickets as seen later in infancy, and certainly is not the same as achondroplasia, at least in origin, although it might simulate it in appearance. The symptoms of achondroplasia or chondrodystrophy have already been described and I need not dwell upon them. Then there is a group of diseases which do not have the symptom-complex of achondroplasia and yet approach it in some respects. Among these are the reported cases where the children are born with short thick bones, as in achondroplasia, but in which there is also a remarkable tendency to multiple fractures. In some instances the vault of the skull has appeared to consist of a multitude of minute plates of bone. There seems to be no name for this except osteogenesis imperfecta, which, of course, is only a method of expressing our ignorance. Cases of this kind have been collected and reported by Chaussier. It is evident that these touch very closely certain

cases of osteopsathyrosis. They are, in fact, instances of this, but of a peculiar kind. I was forced to include them in this category, provisionally, when I reviewed the subject of this latter disease a few years ago.

With regard to what Dr. Jacobi has said concerning the relationship of achondroplasia and fetal rickets, it seems to me very probable that there are certain final results which may be produced by diseases entirely distinct in their origin. It is only as time advances that we have been able to separate affections which have closely simulated each other in appearance, So it may be in this case. However much a certain case may superficially appear to be rickets, yet, if it can be shown that the histological process which constitutes the basis of rickets is absent, then the disease cannot be called this. Now, this has been done in numbers of cases of achondroplasia. There have been found none of the microscopical changes which constitute rickets. Rickets appears to be an inflammatory affection in certain respects. There is an osteitis present. Achondroplasia, on the other hand, seems to be a defect in development. There is no inflammatory process. Although, therefore, we may have produced by each disease dwarf-like and other conditions which are apparently identical, yet in reality they cannot be so, for the pathological processes are different.

DR. HOLT.—I have had a private patient under observation for three years that exhibits these symptoms described and have also had one hospital case of the kind. It seems to me that the clinical picture is something unique and quite distinct from that of rickets.

My patient is now four years old and her height only thirty-two inches. She is a most peculiar looking child and has a large head, circumference twenty-one inches. The bridge of the nose is much sunken, apparently owing to the shortened base of the skull. The fontanelle is still open. She talks well and the mental condition is essentially normal. She began to talk at fifteen months and has progressed steadily. The child lacks the typical bone changes of rickets, the enlargement of the epiphyses and beading of the ribs. It is perhaps of some little interest that thyroid extract was used for nearly two years without any result. A striking feature of the appearance of this child is that the soft parts seem too large for the bones. There are several deep folds of skin between the hip and ankle, as though the skin is too much for the bone.

DR. CHRISTOPHER.—What was the shape of the hands?

DR. HOLT.—The fingers were very broad and very short and nearly of equal length. The growth in the limbs, measuring from the anterior spine to the bottom of the heel, has been exceedingly slow, amounting to only four inches in three years.

DR. GRIFFITH.—I would like to state that in the second case to which I made reference the measurement from the anterior superior spinous process of the ilium to the heel was only about ten inches, while to the top of the head it was between twelve and thirteen inches, showing a distinct shortening of the lower extremities. There were also the curious folds of flesh to which Dr. Holt refers. The appearance of the child was certainly much more suggestive of cretinism than of rickets.

DR. CHRISTOPHER.—I have charge of a family of three such children that I have been watching for three or four years. I had them on thyroid treatment for some time, but without any effect. They are not idiotic, but do not do well at school. Dr. Walker has charge of them for me and he thinks them rachitic, but I cannot think so. The hands are exactly what Dr. Holt describes, the feet short and the legs very short. The appearance is very suggestive of cretinism.

DR. MORSE.—I hardly know how to answer Dr. Jacobi's criticisms unless I read my paper all over again. We seem to differ not only as to opinions but also as to facts. I must refer you to the literature of the subject and ask you to form your own opinions from it. I must speak of certain points, however, as there can be no question as to the truth regarding them. Shortening of the bones of the cranium is not always due to early ossification. It may be due to failure of development. I thought that I satisfactorily explained the relationship between chondrodystrophy and myxedema in my paper. There is no question but that the periosteum as well as the cartilage is involved in rickets. They are both involved, however. In chondrodystrophy the process is entirely different from that in rickets not only as to the time of development but also as to the method of development. The literature to which Dr. Griffith referred was what I skipped in reading my paper. I naturally cannot agree with Dr. Jacobi as to the diagnosis in this case. I never saw a case of rickets in which there was a similar involvement of the shafts of the bones and no enlargement of the epiphyses, as is shown by the X-rays.

THE MANAGEMENT OF RHEUMATIC CHILDREN.

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It is unnecessary before this Society to discuss the symptoms of rheumatism in children. It is sufficient for the purpose of this paper to simply call attention to one of the most marked peculiarities of the disease as it appears in early life. I refer to the frequent distribution of symptoms through months or even years. Several symptoms habitually appear in children which are rarely seen in adult life, for we must class the following in the rheumatic group: arthritis, fibrous nodules, purpura, erythema, chorea, tonsillitis, endocarditis, and pericarditis. These symptoms are, in some cases, massed together, several appearing at the same time. They more frequently, however, appear separately and extend over years of time, as shown in the following case:

H. B., at twelve years of age, gave the following history: An attack of chorea in three successive springs at six, seven, and eight years. "Growing pains" at intervals during these three years. Arthritis of both knees and ankles at nine years, followed in four weeks by chorea. Another attack of arthritis three months later, and chorea again after seven months. Several attacks of tonsillitis during this time. Finally an attack of arthritis with well marked fibrous nodules and endocarditis which left a mitral regurgitant murmur behind. Here we have a rheumatic history requiring six years for its evolution with, perhaps, more to follow.

We are largely indebted to Cheadle for pointing out this peculiarity of rheumatism in children and showing the interrelationship of the various symptoms. It is seldom that so much truth is contained in a single sentence as in the following from Cheadle: "The various manifestations of rheumatism massed together in the case of adults tend to become isolated in the case of children, so that the whole phenomena are distributed over years instead of weeks or months, and the history of a rheumatism may be the history of a whole childhood."

In view of these facts, it is clear that we do not do our full duty by the rheumatic child if we content ourselves with simply treating the various symptoms as we are summoned from time to time. Preventive medicine is a most important part of the work of the modern physician and in rheumatism he has an especially promising field.

The management of the rheumatic child may be considered under four headings: Clothing, exercise, diet, and medication. Before considering these subjects, a word may appropriately be said regarding the etiology of rheumatism. Modern medical opinion seems to tend strongly to the belief that acute articular rheumatism is an infectious disease. This seems a very reasonable proposition, although a specific germ has not as yet been discovered. As a profession, however, we are long passed the stage of thinking that germs alone are sufficient for the production of disease. The soil is necessary as well as the seed, and in most of the infectious diseases we recognize predisposing as well as exciting causes. This is certainly true of rheumatism. Whatever the discoveries of the future may be, we shall no doubt continue to consider certain conditions as predisposing, the prevention of which will be a part of the treatment. Hence, clothing, diet, and hygienic management may very properly receive attention in any discussion of the disease.

The rheumatic child should wear flannel at all seasons, though during the summer it may be of thin texture. Its value as a means of prevention is too well established to be doubted. If it accomplishes nothing more, by diminishing the danger of chill it does much to prevent congestion and inflammation of the throat, an important item in view of the fact that the throat is apparently one of the most frequent portals of entry of the rheumatic infection. Cold and wet feet should be especially avoided.

The exercise and out-door life of rheumatic children should receive peculiar attention. There is a strong tendency on the part of mothers to confine such children to the house too closely and thus render them the more susceptible to cold. On the other hand, there are many days during the winter in which a child of rheumatic tendency should be kept in the house. Days of damp east winds, particularly if the ground be covered by slush or melting snow are especially favorable for the development of rheumatism. Here, as in so many other places, the middle course between too

much coddling and too much exposure is the golden mean to be sought.

The diet of the rheumatic patient was formerly looked upon as of more importance than it is at the present time. When the acid theory was more generally held and when the difference between rheumatism and gout was not as well understood, it was natural that diet should be looked upon as a most potent factor in etiology. We should not, however, go to the other extreme, for it is quite possible, even if the disease be of microbic origin, that a vicious diet may be a predisposing cause. English writers still seem to have almost a morbid fear of meat and nitrogenous matter. This fear extends, in some quarters, even to the use of beef tea, animal broths, and eggs. Many recent writers, particularly in this country, have expressed the opinion, however, that the danger of rheumatism is not increased by the use of nitrogenous diet. The trend of opinion seems to be that restrictions of diet should be rather in the direction of sugar and starch. With this belief I would strongly concur. The rheumatic child is prone to be anemic and I believe that a plain but generous and nourishing diet, which contains some nitrogenous matter, is the best.

In investigating literature quite extensively to ascertain the prevailing opinion regarding diet in rheumatism, I have not found an author who disapproves of milk as the principal diet during an acute attack. The French writers are particularly insistent upon its importance. They deprecate the use even of broths. While milk is unquestionably the best diet for acute rheumatism, I have never seen injury follow the use of properly made broths or plain soups. When the thirst is great, weak lemonade or oatmeal water may be given. As anemia and physical depression develop rapidly, a more general diet should be begun very soon after the temperature approaches the normal and the joint symptoms have subsided. It goes without saying that every case should be carefully studied and the capacity of the patient to take and digest food be made the principal guide in its selection. I would simply advise against keeping the child too long on a weak liquid diet.

As a prophylactic measure proper care of the throat and the removal of adenoid growths and enlarged tonsils must be strongly commended. We may, perhaps, thus close one of the important portals of entry to the rheumatic bacilli.

The preventive treatment of rheumatism does not consist alone

in the use of the measures thus far referred to. Medicinal treatment may be an important element in prevention. We prevent acute attacks of malaria by the repeated administration of small doses of quinin. We prescribe cod-liver oil for more or less continuous use for the children of the so-called strumous tendency. We administer iron for long periods to the anemic. In the same way we may properly prescribe medicine to children of rheumatic tendency. I am convinced that we may do much by the use of the salicylic compounds to prevent acute outbreaks of rheumatism. I have for a long time been in the habit of prescribing for rheumatic children salicylate of soda to be given for a week or two at a time for long periods. Heiman in a recent paper (ARCHIVES OF PEDIATRICS, January, 1901) mentions a similar method. After an attack of the disease, he continues the administration of the salicylates in small doses three times a day for one week of each month for a year or longer. He asserts that Ewart of London, is the only one who has alluded to this method of treatment. I have no desire to take the credit from these two authors, but was somewhat surprised in reading the statement, as I had employed salicylate of soda in this manner for several years. I distinctly remember that Dr. E. LeFevre of New York, in a consultation about five years ago, advised similar treatment. A theory for such use of the salicylates, which seems rational, is that the bacteria or perhaps their toxins may lie dormant in the system for long periods and are destroyed by the continued action of the drug.

Many authors have advised the continuance of treatment for a considerable period after the subsidence of the acute symptoms, and I cannot urge the necessity of this too strongly in the case of children. Whenever there is a recurrence of any of the rheumatic symptoms, I would advise the use of salicylate of soda in doses of from three to five grains, three times daily for one or two weeks of each month, for months at a time. The results of such treatment are particularly satisfactory in those children who suffer from growing pains, who have repeated attacks of tonsillitis, recurring attacks of chorea, or mild arthritis.

Upon beginning the treatment of any disease, it is a wise plan for the practitioner to ask himself what he wishes to accomplish. If he can give a clear-cut and decided answer, he will have done much to render his treatment effective. In the case of acute rheumatism, the answer might well be: 1st. To control the fever.

2nd. To relieve the pain and render the patient comfortable. 3d. To check the arthritis and other local symptoms by neutralizing the effect of the rheumatic poison on the fibrous structures. 4th. To prevent involvement of the serous structures, notably those of the heart.

While the first three of these are highly desirable, they are of little importance as compared with the fourth. Were it not for the danger of cardiac injury, rheumatism, owing to its usual mildness, would be relatively unimportant in pediatric practice.

Two methods are commonly in vogue in the treatment of rheumatism—the alkaline and salicylic. The alkaline treatment proposed by Fuller consists in giving large doses of alkaline salts. It is universally admitted that to be of any value the urine must be kept alkaline in reaction, and the amount of alkali administered must be very great. So firmly was this method established, and so fearful were physicians of the depressing effects of the salicylic compounds, that for many years no author seemed to have the courage to advocate the salicylic method alone. As a matter of fact, however, practitioners were universally using the salicylates and were universally not using Fuller's method. In recent years articles upon the treatment of rheumatism have reflected more accurately the practice of the profession of to-day. Beginning my medical practice in Bellevue Hospital but a few years after the introduction by Maclagan of the salicylic compounds as remedies for acute rheumatism, I saw much of the treatment of that disease by the older method and must say that I do not like it. The alkalies, when given in massive doses, are depressing to the heart and circulation. The treatment is capable of causing as much cardiac depression, it seems to me, as does the salicylic method. Alkalies, moreover, tend strongly to increase the anemia. It seems to me also that the often repeated statement that cardiac complications are less common with the alkali treatment than with the salicylate lacks confirmation, provided that the medication is not stopped too soon. Neither, it seems to me, are relapses more common with the salicylic than with the alkaline treatment, when the treatment is properly persisted in.

Statistics are largely made up from hospital experience. But rheumatism is notably a disease in which hospital experience may prove very misleading when applied to private practice. The hospital patient is much more frequently an anemic, poorly fed, and

badly nourished individual, seen only after the disease is under full headway, than is the private patient. The salicylic compounds act so promptly and efficiently in many cases that the patient is prone to leave his bed and undergo exposure before he can safely do so. Cardiac involvement may thus occur when it would not do so under a treatment which gave less prompt relief of pain. For the same reason relapses are, perhaps, more common, for many patients stop the treatment as soon as they feel better. I am by no means alone in these opinions. Fagge, Strümpel, and Packard have very recently expressed the same beliefs. Fagge asserts very positively that when the salicylates are continued an adequate time after the relief of acute symptoms, relapse is less liable to occur than with other treatment. He also asserts that it is reasonable to expect that any remedy which prevents fresh joints from becoming affected would hinder the development of endocarditis. He believes that this is the fact, though the salicylates do not check endocarditis when it is once established. As to that point, however, it has never been demonstrated that alkalines or any other form of medication have any controlling effect over endocardial inflammation.

I quite agree with Packard, who has written one of the best articles extant on the treatment of rheumatism ("A System of Practical Therapeutics," by H. A. Hare, M.D.), that impressions must, to some extent, take the place of figures in questions so difficult to solve by statistical methods. I agree with him, also, in the belief that the salicylic compounds in shortening the duration of the attack and lessening its severity tend to prevent the occurrence of cardiac complications, which might occur during later weeks were the disease to go on. It is certainly a fact that at the present day, outside of the hospitals, the old dictum of Warren is untrue that the best treatment for rheumatism is "six weeks." Six-weeks' attacks of rheumatism in adults are now far less common than they were twenty-five years ago. I am well aware that attacks of rheumatism in children between five and twelve years are usually mild and evanescent. As they are capable of doing great damage, however, it is our duty to render them, if possible, milder and more evanescent.

Many authorities speak in favor of combining the two methods of treatment and I have made diligent effort to do so. In the case of children, however, I have so frequently found myself in the

position of the proverbial man who tried to sit upon two stools at once that I have abandoned the effort. The alkaline method requires the use of very large doses of alkaline salts at the outset. The salicylic method requires the use of large doses of the salicylate at the outset. Small doses of either are inadequate. The alkalines are very prone to disturb the stomach and the same is unfortunately true of the salicylates and both are depressing to the circulation. The use of both drugs in adequate quantities can be depended upon to upset the stomach of any child. It is far better, it seems to me, to select either one or the other and hold to it as the chief treatment. If the treatment by salicylates is selected, after the doses of the first few days have been reduced, an alkali may be added to advantage, but the stomach should be carefully watched. I often administer bicarbonate of soda with the milk, more, however, for digestive purposes than with the idea of relieving the rheumatism.

It is wise at the outset to begin the treatment of rheumatism with a cathartic, and calomel in small repeated doses has particular advantages. The condition of the bowels should be watched throughout the course of the disease for their locking up is almost invariably followed by increase of the symptoms. Saline cathartics have many advantages but their overuse is to be avoided in children as they tend to increase the anemia.

It is important to begin the administration of a salicylate at the earliest possible moment. The salicylate of soda, all things considered, has been in my hands the most satisfactory preparation. It may be given in doses of five grains every three hours to a child of six years, forty grains per day. After the third day, if the disease seems to be under control, the interval may be increased. If the case is a severe one, or if no improvement is seen after the first day, the interval should be reduced to two hours, so that sixty grains are taken in one day. During the first two or three days the doses should be given at regular intervals, day and night. They are usually better tolerated if not taken on an empty stomach. Hence, it is well to give the nourishment at the same interval, and administer the salicylate after it. As a vehicle, I often use an essence of elixir of pepsin which is palatable and apparently counteracts a tendency to vomit.

Salophen is one of the best substitutes for the soda salt, but the expense is sometimes an objection. Salol, it seems to me, is

rarely available in acute rheumatism, particularly in children. The oil of wintergreen is, in my experience, more depressing than the salicylates, and after a few doses is often refused by children. Salicin is difficult to administer to young patients. It may be given in doses of three grains every three hours at six years.

Hyperpyrexia is extremely rare in the rheumatic attacks of children. When it does occur it should be treated with cold baths or cold packs with, perhaps, small doses of phenacetin well guarded by stimulants. Stimulants are otherwise very rarely required by children with rheumatism.

The rapid development of anemia is one of the most marked characteristics of rheumatism in children. I begin the administration of iron, therefore, at the earliest possible moment. The time that moment arrives has, however, been a question of much discussion. It was the teaching of Prof. Loomis, under whom I served as house physician, that iron should be commenced as soon as the temperature ranged below 100°. I have rarely seen bad results follow such use. The disease is so prone to relapse that it is impossible to say in most cases whether it is due to this or that cause. For children convalescing from rheumatism, I have long used Basham's mixture (solution of iron and ammonium acetate) which admirably fulfills the requirements. It is palatable and well tolerated by the stomach, while its diuretic action is extremely favorable.

These children are so prone to be anemic that I feel impelled to give a generous diet as soon as it is possible. In some cases I have not hesitated to give beef juice and even a little meat very soon after the disappearance of the symptoms. The sooner we can restore the nutrition, the less liability will there be to relapse. There are, moreover, some cases of rheumatism occurring in anemic, poorly nourished children in which no improvement can be secured until iron and cod-liver oil are administered. The sooner we can improve the nutrition, the sooner we control the disease, and the less we see of relapses and complications.

Local treatment may be effective in relieving the pain and in some cases, perhaps, in controlling the severity of the arthritis. Every rheumatic joint should be protected from the air by a flannel bandage or by cotton wool. A light splint of basswood or pasteboard will often give great relief. If but a few joints are in-

volved, they may thus be immobilized and the patient can be moved with comparative comfort. The arthritis in children is so transient that it seems useless to apply plaster of Paris, or even a starch bandage. Heat in my experience has been more acceptable to most patients than cold. I rarely use watery applications, though a lead-and-opium wash may give some relief. Unless carefully applied, by their evaporation, they cause more pain than they relieve. I have used oil of wintergreen and various liniments locally, but have been unable to see any material results. The odor of these preparations, particularly wintergreen, becomes very objectionable to some children. Blisters should never be used in young children, even in subacute and chronic cases.

Children suffering from any rheumatic manifestation, even chorea, if it be severe should be put to bed and kept there absolutely until every symptom has disappeared. I feel like speaking very strongly upon this point for it is one upon which the average practitioner frequently errs. Rest in bed will not always prevent cardiac involvement, but will do more to that end than will any other means we possess. Without such rest, it is impossible to control certain cases even when subacute. The symptoms will continue for days and will repeatedly recur. The patient should be placed between blankets instead of sheets and a light flannel jacket should be worn. This will enable the child to sit up in bed without danger of exposure and he will thus be less discontented during the days when the symptoms are slight.

We cannot always prevent cardiac disease in our young patients of rheumatic tendency. By proper diet, clothing, exercise, hygienic management, and perhaps in some cases by medication we may do much to prevent acute attacks of rheumatism. Such an attack having occurred, we may still do much to prevent cardiac involvement by diet, rest in bed, protection from exposure, and medicinal treatment. The attack having been controlled, we may lessen the danger of relapse by continuing all the precautions, improving the nutrition, and persisting in the medication. In closing, I would again urge the vital importance of nutrition-improving treatment, and the folly of relying wholly on specific medication in the rheumatism of children.

DISCUSSION.

DR. CHAPIN.—I was very glad that Dr. Crandall brought up this subject, and treated it in such a broad way, because I believe that physicians, as a class, do not always do their whole duty in impressing families with the importance of treatment in rheumatism. A child with rheumatism is a child that needs, not a week's attention, but months and perhaps years of care. If we could give that attention we would ward off a large number of chronic valvular diseases later in life.

I believe that a large proportion of valvular diseases begin in childhood. We see children with these pains occurring every few months and a little later on we find them with chronic valvular disease. In children the symptoms of endocarditis are so slight that they are passed over. The complaint of pain is slight, or not at all, yet there is the hopeful time for treatment. There is soft heart muscle but the child is allowed to run around until finally we have dilatation, and incompetency of the valves and permanent disease. Owing to the fact that the arteries are so healthy in early life there may be no symptoms at all. The effect of the arteries on the circulation we often overlook; they are elastic and healthy and help out the heart. With middle age the other symptoms come on. Patients do not know how long they have had the trouble, but in the majority of cases it has begun in childhood. If we would follow out Dr. Crandall's ideas we might prevent many of these cases developing serious symptoms. The principal thing, perhaps, is absolute rest. I agree with him that we must keep on treating these children.

With reference to the modern idea that there is a microbic origin for rheumatism, I am not sure, but I believe that that theory is best in following a disease that gives us the best results in practice, and I believe the best results are obtained by following the old theory of an acid condition. By preventing the acid from forming, and by acting upon the skin and intestinal tract to rid the system of the acid I get the best results. With reference to the salicylate of soda I agree that it should be given for a long time in small doses, but would call attention to one peculiarity in its action and that is, an occasional purpura following its free administration. We should be on the lookout for that, and should also bear in mind that the drug is depressant to a certain extent, but by giving it in small doses, and at the same time treating the digestive tract carefully and acting upon the skin we can do a great deal with it to avoid the occurrence of endocarditis.

DR. JACOBI.—Everybody knows what I am going to say; that prevention is better than cure. Get the children used to cold water in whatever shape they bear it best. Rub them well and get up a good circulation. Attend to their throats. Have hypertrophied tonsils removed. I find a great many such throats and

find that the people have been consoled with the expectation that in a number of years, at seven or thirteen, they will be all right. In the meantime rheumatism develops, or other infections take place.

DR. CARR.—One thing I have found of service in these cases is the employment of the fats; cod-liver oil is always the best, but butter and cream are valuable. Frequently iron is not well borne and yet the child seems to need something more than a general so-called tonic. By the use of cod-liver oil, in doses according to the digestive strength of the child, much more will be gained than with the use of the hypophosphites, arsenic, or substitutes for iron. We can hardly be too emphatic in endorsing what Dr. Jacobi has said regarding the tonsils.

DR. PACKARD.—In the course of his remarks Dr. Crandall mentioned, incidentally, the asserted depressing effect of the salicylates upon the heart. It seems to me that too much importance is by some attached to this effect, and I do not believe that this depressing action, even if it is as marked as some hold, is of much disadvantage. In rheumatic endocarditis our patients do not die nor do they seriously suffer because their hearts are depressed. In these cases I believe it to be eminently desirable to lower the activity of the heart, and it has always seemed to me that in cases of acute endocarditis, digitalis, which is sometimes strongly urged in this condition, should only be employed where after careful consideration it is determined that its use is necessary to save life. I should think, therefore, that if the salicylate is depressing to the cardiac muscle it is rather an advantage than otherwise.

DR. KOPLIK.—As to the removal of the tonsils in these cases I would like to call attention to the fact that before any operation upon the tonsils, or adenoids, is performed on these little ones we should be certain that endocarditis, as an acute process, has come to an end. I have seen a case in which the physician, though he knew that endocarditis was recent, advised operation on the tonsils and adenoids. The tonsils and adenoids were removed; within a few days there was chill—although the operation was done under the proper precautions as to asepsis—a new attack of endocarditis set in and caused death in a very short time. Such cases are extremely exceptional, of course, but should warn us to wait for quiescence of the cardiac condition even though the tonsils and adenoids are very marked.

DR. WILSON.—There are two or three things I would like to say in this connection. The first is this, that perhaps to me the most important point made by Dr. Crandall was in the title of his paper, in which he uses the words "rheumatic children." It leads up to a proper conception of the predisposition to rheumatic

fever and enables us to see that there are important generalizations to be made in regard to the whole subject. Some time ago Dr. Packard emphasized the importance of the relationship between tonsillar angina and rheumatic fever. In some individuals, by reason probably of anatomical peculiarities, there is an especial liability to infection.

A mild angina may be the only result. If a general rheumatic infection takes place certain tissues only may react to it. In some cases there are joint manifestations, in others endo- or pericarditis or the two combined. In a small proportion of cases a rheumatic pleurisy may arise or rheumatic peritonitis, or we may have meningitis—so-called cerebral rheumatism. These facts are trite and familiar, but their consideration and the recognition of the part played by the various tissues in rheumatic fever leads to a broad conception of the disease. A trifling angina may be followed by an equally trifling joint affection or none at all, or the earliest manifestations may be endocarditis so slight as to attract no attention. The child becomes anemic, fails in health, loses appetite and upon investigation is found to be suffering from endocarditis. The joint affection may be overlooked or regarded as a manifestation of "growing pains." The febrile movement also is often trifling. Such attacks recur from time to time, to be succeeded after a while by an attack of well-marked rheumatic fever. When such a child reaches the age of puberty it will be found very often to have signs of chronic valvular trouble.

As to the continuous administration of the salicylates I find myself disposed to be at variance with the reader of the paper. Perhaps I have not developed the proper appreciation of drugs. I would prefer to await the development of distinct rheumatic symptoms before using the salicylates. I am distinctly at variance with Dr. Packard as to the advantage of depressing the action of the heart. I believe that the proper method of easing up the work of the heart is to reduce the height of the column of blood which it has to lift at every stroke and thus minimize its work. This is best accomplished by keeping the patient in the horizontal posture for several weeks after the acute symptoms have disappeared.

I agree with Dr. Jacobi that general hygiene, particularly that of the skin and throat, constitutes our very best prophylactic treatment.

DR. BLACKADER.—Digitalis is rarely called for in any acute affection of the heart. I believe in the necessity in the rheumatic affections of children for the administration of the salicylates for some time after the symptoms have subsided. As to the use of the potassium salts, I think that very few of us would feel justified in pushing their administration to the extent that Fuller did, but the potassium salts may be employed in smaller doses, I think with

good effect, stimulating diuresis and assisting in the elimination of the toxins. I question whether we have any right to infer that because quinin will ward off malaria, salicylic acid given occasionally in small doses will ward off the development of rheumatism. I must confess that my experience leads me to place more dependence upon increasing the resisting powers in the child by treatment of its general condition, securing a vigorous reacting power in the skin, and a healthy condition of the tonsils. I do not think that we yet know enough about these specific drugs to use them in a prophylactic way. In every other respect I agree with Dr. Crandall.

DR. PACKARD.—I would like to correct what I think is a misunderstanding of what I meant to say in my remarks. I did not mean that I would give the salicylate for the purpose of depressing the heart, but that it is better not to give a drug to stimulate the heart.

DR. CRANDALL.—I think from my rather prolonged discussion of other points that there has been a misunderstanding as to the amount and frequency of using the salicylate that I advocated. I said only at *intervals*—one week out of three or four, and then only where there is decided tendency to recurrence, or in a case like one I saw a week ago in a child of nineteen months, where there was decided articular rheumatism. The arthritis was as distinct as in any adult. In twenty-four hours both knees, ankles, and wrists were involved. The mother of the child was also rheumatic. One could hardly expect that mere hygienic management would prevent that child from having additional attacks. I believe in such a case that small doses of the salicylate of soda, two or three times a day, for a week at a time, is good treatment. I would not advocate giving it continuously.

THE USE OF THE TERM "ENANTHEM."

BY F. FORCHHEIMER, M.D.

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At the Cincinnati meeting of this Society I had the honor of presenting a paper on the "Enanthem of German Measles." Some doubts were expressed in the discussion as to the propriety of using the term "enanthem." The object of this paper is to dispel these doubts and, at the same time, to ask your co-operation in assigning to this term a proper place in our nomenclature.

It is difficult to say by whom the term was first employed; I find it first used by the French (Cadet de Gassicourt, 1882). After this it was employed by the Germans. In Eulenburg's Real Encyclopedie, edition of 1880, the word is not found; but in the one of 1886 it occurs with a definition. At the present time the term has been universally adopted by the French authors; no modern description of the acute exanthemata exists in which it is not used. In Germany also, the word is being made use of more and more constantly, although Villaret's "Handwoerterbuch der Gesammeten Medizin" (1899) prefixes its definition by calling enanthem "ein seltenes Wort" (a rare word). Notwithstanding this, we find in the latest of the larger works on practice, Schwalbe and Ebstein, Vol. V. (1901), the period of invasion in rubella spoken of as the "Stadium enanthematicum sive prodromorum." In addition to those mentioned before I find the term in the Dictionaire Encyclopédique des Sciences Médicales (1887) and in Littré. In English it is found in the following dictionaries: Dunglison and Foster's medical dictionaries; the Standard; the Century and the Imperial as enanthema, in Worcester as enanthesis, always with the definition which seems the correct one.

In recommending a new term made up of two Greek words, although it may have been accepted by many other authors, it is well to go into its derivation, for, after all, we physicians do not want to be looked upon as outer barbarians. We already have a suffi-

ciency of terms that are etymologically sinful. That "enanthem" has been accepted by the Germans and the French is distinctly in its favor, as they are slow to act in this direction; as expressed by Hofmann, in connection with a word made of a Latin noun having joined to it a Greek ending, some of them do not wish to hurt their conscience which has been developed by their training at the gymnasium. It may be said to begin with, that the word ἐνάνθημα does not occur in the Greek authors; it is not found in either modern or ancient Greek; so that its meaning must be worked out from another word which is constantly employed, *i.e.*, ἐξάνθημα.

Concerning the propriety of this word there can be no doubt, as Hippocrates uses it constantly in his aphorisms; I do not find it in Hesychius, but quite a paragraph is devoted to it in Stephanos. The derivation of this word is the preposition and ἄνθημα from the Greek root $A\theta$, which means to blossom. Stephanos defines this word ἄνθημα, τό as *florum emissio; florium productio, vel floris: efflorescentia, si ita dicis posset, ipsus florendi actio.* Ἐνάνθημα would then mean a florescentia within, in contradistinction to a florescentia without, ἐξάνθημα. "Ἀνθημα" is, as far as I can find, a term which, when used at all, is simply used for its botanical meaning, while ἐξάνθημα is a purely medical term. In looking over the various definitions given for this latter word the conclusion is inevitable that anything appearing upon the skin may be considered an exanthem; thus blisters, pustules, scabs and various kinds of tumors (Stephanos) papules and mucous tumors (Pliny). The modern writers still divide as to the definition of the term whether it is to be used as the external manifestation of the eruptive fevers only, or other eruptions as well. For our purpose it is not necessary to go into this discussion, only in so far as to verify the fact than an exanthem is looked upon by various authors in various ways. Some claim that an enanthem is an exanthem upon the mucous membrane, the definition then resolving itself to the definition of an exanthem. I have serious doubts as to the propriety of confining the term "enanthem" to the specific manifestations of the eruptive fevers; it would certainly not be wise to do this until a majority of authors should unite in giving this purely technical meaning to this term.

A second class of definitions of the term "enanthem" is the one represented by Villaret, "all efflorescences occurring on the in-

ner surface of natural cavities, for instance, the nose, the throat, the mouth, etc., without pathological distinction," corresponding then with the view just held, except in that it seems too broad to be applied to the acute exanthemata. It seems to me that when the term "enanthem" is applied to the acute exanthemata it should be held as meaning only the specific eruption of the especial form or forms of acute exanthemata that are found upon the patient. In measles, for instance, it is not uncommon to find stomatitis herpetica, yet this cannot be called the enanthem of measles. On the other hand, there are certain changes produced by the acute exanthemata upon the mucous membrane, the streptococcus angina scarlatinæ, especially which in the present state of our knowledge it would be doubtful whether they could be rightly held to be included under the heading of the enanthem of scarlatina. If Class's work becomes generally verified then this doubt disappears. The enanthem of an eruptive fever would then be the various manifestations upon mucous membranes of the cause of this eruptive fever, or such manifestations as occur in the vast majority of cases. But it will be found as these enanthems are more carefully studied that it is not a manifestation that localizes itself to one mucous membrane, or a part of it, but as a rule is extensive; in measles, the soft and hard palate, the cheeks, the lips, the tongue, the greater part of the respiratory mucous membrane; in scarlatina, the soft and hard palate, the cheeks, the tongue, the tonsils, and the stomach and intestines.

The question that arises finally is, Why should we use the term "enanthem" at all? Etymologically there are no objections which can be raised to its use. It is extensively adopted by writers of other nations, and we ought to do all we can to make the language of science international. Time is saved by using one word instead of many—as "the appearance upon the mucous membrane," instead of "enanthem." Any objections that can be raised to the want of precision in definition must fall away, in that when the term is used as applied to the eruptive fevers the connection will always preclude any doubt as to its meaning.

DISCUSSION.

DR. CAILLÉ.—I was one of those who objected to the use of the term enanthem. An eruption or exanthem is a perfectly definite term and conveys a clear meaning whether the eruption

be located on skin or mucosa, and I do not think we should complicate matters by adding to our nomenclature in this instance.

DR. GRIFFITH.—Dr. Forchheimer has shown that this is not a new term. I remember seeing the term used in German books before I was a graduate.

DR. WILSON.—Dr. Forchheimer's communication strikes me with some surprise, because for a number of years in my teaching I have used the term enanthem in exactly the sense the doctor describes it as indicating the specific eruption on the mucous membrane in contradistinction to the eruption upon the skin, and I am pleased and gratified to have him call attention in this way to the matter, but it seems a work of supererogation.

DR. ROTCH.—I do not see why if a term is incorrect we should go on using it, or if a term is correct why we should not adopt it just because it multiplies terms. The sooner we all get to work and eradicate the old terms, the better. We should all use our influence to bring the right terms into general use. If I am using an incorrect term I am very glad to be corrected. What is needed is uniformity.

DR. SEIBERT.—The prefix ex implies something that is within and goes out; the en implies something that is outside and goes in. It is the same eruption, whether on the mucous membrane or on the skin, the same pathological condition, and the same term should be used in both instances.

DR. FORCHHEIMER.—I am delighted to hear that Dr. Wilson is using the term. You can include all the eruptions on mucous membranes calling them enanthem, but in our English text books we do not find this term. The only man who has ever used the term in this country, so far as I know, in a text book, was Dr. Whitaker, my colleague. If the term is of any use we should all help in getting it into the literature.

DR. CHRISTOPHER.—Would the word eruption be proper to use? If we wish to distinguish between mucous membrane and skin can we speak, for instance, of the eruption in the mouth?

DR. FORCHHEIMER.—An eruption on the mucous membrane is an enanthem; it saves us just this phrase.

REPORT OF A CASE OF EXTREME ENLARGEMENT OF THE SPLEEN WITH ANEMIA.

BY SAMUEL MCC. HAMILL, M.D.,

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The patient whose case I report first came under my care in March, 1897, at the age of six years. A few weeks later I presented him to the Philadelphia Pediatric Society with the following report, revised only in so far as it relates to the family history. At the time of his death I found out what I had not discovered before, that he was the illegitimate son of a woman whom I had been led to believe was his sister. No information regarding the health and habits of the father could be obtained. The mother is living and in good health, without any history or manifestations of syphilis. No member of the mother's family has had any disease similar to the patient's. All of his uncles and aunts (four in number) are living and well. His grandmother died of pneumonia in February, 1900. His grandfather is living and well. The patient has never had any of the early manifestations of congenital syphilis and gives no history of malaria. His only illness has been an attack of epigastric pain of several weeks' duration, which occurred about two years ago. His abdomen was not examined then or at any previous time. For several years he has had frequent and sometimes profuse epistaxis. His skin has had a dusky hue since birth; it has never been pigmented.

Eight or nine weeks prior to coming under my notice he developed constant epigastric pain, which increased shortly after eating. Following this by one week he began to have constant severe pain in the lumbar region. A week later he had five hemorrhages from the stomach in the course of a single day; about a cupful of dark clotted blood was vomited, and on the same day he passed some blood from the lower bowel. He was pale and very weak following this and was confined to bed for a week. Immediately following the hemorrhages the pain in the back ceased.

The epigastric pain continued for some time and he has vomited once since this attack, but there was no blood in the vomitus. He has gradually regained his strength, appetite and color, and within the past two weeks the pain has ceased entirely. He did not seem to lose flesh in the attack. His bowels are regular and have been since birth. He has never vomited blood before, nor passed blood in his stools, nor from his bladder. No urinary symptoms of any kind have ever been noted. His body temperature has been slightly elevated in the few times he has been seen, ranging from 99° F. to 100½° F., and his grandmother thinks he has mild fever quite often. He has always been a very active boy, engaging in all the violent sports without any apparent discomfort or disadvantage to himself.

THE PHYSICAL EXAMINATION shows a fairly well-developed boy, somewhat under size, anemic, and the skin having a rather dusky hue. The most striking thing about his appearance is the marked prominence of his abdomen, especially in its left half. This condition his grandmother thinks has existed practically since birth, at least she has long been conscious of it and cannot fix upon the time of its beginning. The superficial veins of the thorax and abdomen are unduly prominent. Palpation of the abdomen shows a huge, firm, smooth mass, occupying the entire left upper quadrant, extending some distance below the transverse umbilical line and slightly to the right of the median line in the umbilical region. The mass has well-defined edges and a distinct notch in the right border. The percussion note is dull over the area described—the dullness extending to the vertebral column posteriorly and superiorly to the seventh rib in the anterior, and to the eighth rib in the posterior axillary line. The mass moves obliquely downward on inspiration and can be moved about quite readily by the hand.

Palpation of the right half of the abdomen shows the liver to extend slightly beyond the margin of the ribs. Its upper border corresponds to the fourth rib in the midclavicular line. The remainder of the abdomen is rather tense. The intestines are evidently pushed over into the lower right abdomen, where there is decided tympany.

The lungs are normal. The apex beat of the heart is in the fourth space just outside the midclavicular line. There is a soft systolic murmur heard at the apex over the body of the heart,

and in the pulmonary area, and there is a venous hum in the neck. The lymph nodes at the angle of the jaw, the post-cervical, the axillary and the inguinal lymph nodes are slightly enlarged; the epitrochlear and suboccipital nodes cannot be felt. The head is well-shaped; the legs are straight. There are no deformities of the thorax save a slight costochondral thickening. The examination of the urine for sugar, albumin, casts and tubercle bacilli was negative.

Dr. A. E. Taylor, Associate in Clinical Medicine in the Pepper Laboratory, kindly made an examination of the blood for me on the 24th of March, 1897, with the following result: H., 40 per cent., erythrocytes, 3,365,000; leukocytes, 5,200; differential count, polynuclear forms, 50.4 per cent.; mononuclears, 7 per cent.; lymphocytes, 42 per cent.; eosinophiles, 6 per cent. The only change shown in a second examination, made on the 13th of April, was a slight increase in the red corpuscles and an advance of 10 per cent. in the hemoglobin.

After the above notes were recorded, I did not see the patient again until the 27th of October, 1899, an interval of nineteen months. During this time he had been, and at this date was entirely, well, and only returned because of partial deafness, which had dated from the 4th of July immediately preceding. A physical examination was made, however, and the following notes recorded: The general appearance is much the same as at the previous examination; he has grown rapidly and seems normal in height for a boy of nine years.

ABDOMINAL EXAMINATION: The abdomen is very prominent, especially in its left half; the lateral diameter is greatly increased, making the costal angle very obtuse, the slant on the left being nearly parallel to the plane of the chest. There is marked fullness of the superficial veins of the chest and abdomen. In the left upper quadrant and extending into the lower left quadrant beyond the crest of the ilium and anteriorly one and one-half inches beyond the median line, there is the same firm, smooth mass with the same well-defined edges, evidently an enlarged spleen. By pressure on this mass from right to left, the right border can be pushed to three inches to the left of the median line. Pressure from above downward causes the lower border to descend several inches below the point described. By applying pressure from below upward, this same border can be elevated above the trans-

verse umbilical line. The upper border of dullness in the position first described corresponded in the anterior axillary line to the seventh rib, and in the posterior axillary to the eighth rib. This boundary changes in correspondence to the changes in position described. The area of liver dullness is not increased, and its edges do not extend beyond the costal margin; the rest of the abdomen is tympanitic; there is no tenderness.

Heart: The apex beat is visible and palpable in the fourth space just within the midclavicular line. The area of dullness begins above at the second rib, on the right at the left edge of the sternum and on the left in the midclavicular line. There is a soft systolic murmur best heard at the apex, heard faintly at the fourth costal cartilage and in the pulmonary area; it is not transmitted to the axilla. The lung examination is negative.

A complete blood examination was made at this time by Dr. C. Y. White, but unfortunately the slip containing the exact percentages was mislaid; it showed, however, a higher percentage of H. than before; the red cells were increased, and the whites decreased. The differential count showed a smaller number of lymphocytes, and a greater number of polymorphonuclears.

Practically the only change noted since the last examination was some increase in the mobility of the spleen.

On the 15th of January, 1900, about three months later, he consulted me on account of a cough which he had had for four weeks. The following notes were recorded. Cough especially severe at night; it is loose, but unaccompanied by expectoration. He has quite marked dyspnea, which is increased when he lies on his back or on his right side.

PHYSICAL EXAMINATION: The abdomen is more distended; the splenic mass is the same in size, position and mobility. There is some dullness in the flanks and a suggestion of fluctuation, due possibly to a slight effusion.

Lungs: The examination shows bilateral dullness posteriorly, which is probably due to the enlarged spleen on the left, and the pushed up liver on the right. There are some moist râles over the lower chest, posteriorly, especially on the right side. There is no bronchial breathing and the vocal resonance and the tactile fremitus are normal. There is some pigmentation of the skin over the forehead toward the temporal regions. The bowels are regular. The amount of urine voided is less than normal.

mediately began to vomit; the quantity of blood lost was thought to have been quite as much as on the previous day. His condition was such following this hemorrhage that he had to be actively stimulated before consciousness was restored. When seen at ten o'clock on the following morning he had a small, irregular pulse, beating 120 to the minute. His heart sounds were weak and muffled. His pallor was extreme; he was yawning and sighing persistently.

His abdomen was not palpated at this time, but it looked much smaller than it had been, a fact which his attendants had noted. He had three tarry stools on the second day after this attack. His condition improved somewhat until, on the morning of the 27th, he expressed himself as feeling much better; he talked a great deal and asked to have the paper read to him. About ten o'clock, while in the act of drinking a glass of milk, he fell back pale and unconscious, and vomited a mouthful of blood. He had several general convulsions, developed Cheyne-Stokes' breathing, and died in three-quarters of an hour without regaining consciousness. His abdomen, examined one-half hour before death, was flat, and the spleen was greatly reduced in size, extending only about three inches beyond the costal margin.

THE AUTOPSY, restricted to the abdomen by the request of the mother, was made six hours after death. Emaciation was extreme; the skin was of a lemon hue; postmortem rigidity was moderate; the body had retained an unusual degree of heat. An incision was made from about the second costal cartilage to the pubic bone. The skin and subcutaneous tissues were very tough, and there was an unusual deposit of bright yellow subcutaneous fat. The parietal peritoneum was much thickened, and had the appearance of an aponeurosis. There was evident to the view only the coils of the small intestines and a small portion of the spleen, showing just below the margin of the ribs in the upper left quadrant. The omentum was exceedingly fatty, the fat having the same peculiar color as that already described. The intestines had a pearly appearance, their peritoneal covering being greatly thickened. The small intestine was not adherent except in the region of the spleen. The appearance of the mesentery was most striking. It was infiltrated throughout with a dense sheet of light yellow fat which was about three-sixteenths of an inch in thickness. There was some enlargement of the mesenteric lymph nodes ranging in

size from a pea to a lima bean. They were embedded in the fat. The stomach was distended and full of liquid. On removing the intestines the mesentery cut very much, as did the abdominal wall; it was richly supplied with blood vessels. The transverse colon was very adherent to the spleen, the kidneys, the upper portion of the small intestine and the liver. The stomach contained about a pint of liquid and clotted blood. There was a great deal of mucus adherent to its walls. When this was washed away the mucous membrane was found to be pale; there were no areas of congestion; all of the vessels were empty; there was no evidence of ulceration or thickening, and there was no fullness or varicosity of the esophageal veins. The stomach wall was thickened, due apparently to thickening of the serous coat. The walls of the intestines were very much thickened, due again to thickening of the serous coat. In the rectum and sigmoid flexures, there were hard fecal masses; in the large and small intestines there was a considerable quantity of blackish rather pasty fecal matter, evidently altered blood. The spleen was adherent especially in its attachment to the stomach and to the bend of the colon. There had evidently been some inflammation of the peritoneum in this region at some time, as the firmness of the adhesions made the removal of the spleen difficult. It was seven inches long by four inches wide; its capsule was extremely thickened, giving it a whitish appearance. On section it was pale red in color. There were numerous grayish spots and streaks throughout, marking areas of proliferation of the fibrous tissues. There were also many very small, brownish yellow spots which were shown by the microscope to be areas of old hemorrhage. The pancreas was embedded in fat and very adherent to the stomach and intestines. It was very hard and enlarged, especially the head of the pancreas. The gall-bladder was free; the liver was considerably enlarged, of rather peculiar shape, being especially thick in its antero-posterior diameter. Its surface was pale; it was very firm, very pale in color and rather gritty on section. The kidneys were larger than the adult kidney. The left suprarenal was overlooked, the right was removed attached to the kidney; it was much embedded in fat, not enlarged and apparently normal. The kidneys on section were found to be exceedingly pale. The cortex was from three-eighths to one-half an inch in thickness. The substance of the kidney was very firm, the pelvis was empty. The bladder con-

tained a moderate amount of blood-free urine; the peritoneum overlying its abdominal surface was thickened.

Thorax: The thorax was examined by the retraction of a portion of the ribs. The lungs were found to be entirely free, very pale and slightly emphysematous. There was an unusual amount of mediastinal fat of the same peculiar yellow color as that above referred to. Occupying the position of the thymus there was a mass about three inches long by one and one-half inches wide, and one-quarter of an inch in thickness, which had in a dim light the appearance of fat. There was some enlargement of the bronchial nodes. The heart contained no blood; the walls of the left ventricle were rather thickened; the right ventricle was dilated and its walls very thin. The valves were normal throughout. The heart muscle was relatively less pale than the other organs of the body. A section of the tibia was removed. The bone marrow was pinkish in color and seemed to contain less fat than normal. There was very little blood in any portion of the body; that which was present was very pale and not clotted.

The histological examination was kindly made for me by Dr. C. Y. White, Assistant Director of the Pepper Laboratory of Clinical Medicine, who reported as follows:—

"The heart shows nothing abnormal. The liver cells are slightly cloudy, otherwise normal. The biliary ducts are surrounded by much connective tissue; in some areas this is from five to seven times more than normal, otherwise there is no increase in the connective tissue in the organ. The kidneys show slight cloudy swelling. The thymus gland is normal.

The Lymph Nodes: (1) Mesenteric. The capsule is not increased in thickness. The lymph follicles are prominent, being increased in size and number. The blood supply of these nodes is rich, in places it seems excessive; in still other areas there are distinct hemorrhages. Many of the lymph nodes contain fat cells, some of the lymphoid tissue being arranged in crescentic form around collections of fatty tissue. (2) Bronchial. The connective tissue is increased in the capsule and trabeculæ of the lymph nodes; they show moderate congestion.

The Pancreas: The cells look small, and there is great increase in the connective tissue around the ducts, even more than in the liver.

The Spleen: Stained with eosin and hemotoxylon; there is

enormous increase in its connective tissue. The capsule is four to six times its normal thickness; from the capsule the fibrous tissue dips into the splenic substance as a coarse network forming the trabeculæ. Directly under the capsule this network is very prominent, and encloses in its meshes numerous small round cells. Throughout the whole organ the trabeculæ are greatly increased in thickness and the splenic pulp is reduced. In this overgrowth of connective tissue the elastic fibers are very prominent and seem to be increased. The trabeculæ are well supplied with blood-vessels. Large hemorrhages are noted in several areas of this new tissue. The blood corpuscles are sharply defined in these hemorrhages and seem to be of recent exudation. In other areas there are collections of irregular clumps of fine and coarse granular pigment of light yellow to a yellowish brown color. Occasionally this pigment forms distinct plugs in the channel of the fibrous tissue. The pigment gives a very pronounced iron reaction, and is in all probability the remains of an old hemorrhage. The splenic pulp shows a marked reduction in the number of the Malpighian bodies, while those present are undersized. The rest of the pulp tissue contains many red blood corpuscles which increase in number as the Malpighian bodies are approached.

The Bone Marrow is reddened and its fatty elements are decreased. A cross section of a rib shows that the fatty tissue has been largely replaced by cellular elements which consist of large mononuclear cells (large neutrophilic myelocytes), small lymphocytes, and an increase in the number of eosinophiles. There are only a few nucleated red cells and polymorphonuclear leukocytes."

The features of this case which seem to me to be of particular interest are: (1) The great size of the spleen, extending down to within a few inches of the pubic bone.

(2) The early beginning of the splenic enlargement.

The fact that when the boy was first seen at the age of six years he had a spleen almost as large as at the time of death; the history of abdominal pain of several weeks' duration two years prior to this; the occurrence of frequent epistaxis at an even earlier period, and the statement of the grandmother that she failed to remember the time when he did not have an enlarged abdomen, are all facts which would seem to fix the beginning of this condition in infancy or early childhood.

(3) The series of profuse hemorrhages.

(4) The length of duration with so little influence upon the health of the patient. The only time that he ever suffered any serious inconvenience was at the time of the occurrence of the hemorrhages.

(5) The attack of extensive abdominal ascites, its entire disappearance and the subsequent excellent health of the patient.

(6) The mild degree and peculiar type of anemia. Chlorotic anemia with leucopenia.

There were also certain interesting conditions discovered at the autopsy, for which I have no adequate explanation; they were:

(1) The presence of so much fat, of such a peculiar bright yellow color, and especially its extensive deposit in the mesentery.

(2) The apparent general hyperplasia of the fibrous tissues.

(3) The pearly appearance of the peritoneum, especially of the visceral peritoneum. This was evidently a thickening of the fibrous elements of the peritoneum, and probably a part of the general process of hyperplasia of the fibrous tissues, and not the result of an inflammatory process limited to the peritoneum.

(4) The peculiar microscopic evidences of old hemorrhage into the spleen as described by Dr. White's report, and as seen under the microscope. And (5) The condition of the mesenteric lymph nodes. This latter is especially interesting in connection with the recent article by Warthin on "Hemolymph Glands of the Sheep and Goat," published in the May number of the *Journal for Medical Research*, in which he reports the results of a study of the lymph nodes at varying intervals, following the operation of splenectomy in these animals. In the July number of this journal for 1901, he advanced the hypothesis that under certain conditions the lymph nodes are capable of compensating for the spleen. This hypothesis "was based partly upon the general similarity in structure of hemolymph nodes and spleen, their common function of hemotolysis and the occurrence of transition forms resembling accessory spleens, but chiefly upon the autopsy findings in a case of splenic anemia, in which there was throughout the mesentery fat a new formation of hemolymph nodes resembling splenic tissue." The work recorded in his recent article was undertaken to confirm this theory.

The conclusions he reaches are as follows:

(1) "After total splenectomy in the sheep there is no evidence

of regeneration of the primitive spleen or of the new formation of splenic tissue."

(2) "The structural changes following splenectomy are, hyperplasia of existing lymph tissues, transformation of hemolymph nodes into ordinary lymphatic glands, and a new formation of hemolymph nodes out of" (this expression is misleading, and the author's meaning would seem to be better expressed by substituting the word in for "out of")* "lobules of fat tissue, and a later proliferation of the red marrow."

(3) "There is no evidence of red blood cells in the lymph nodes after splenectomy."

(4) "The function of hemolysis is taken up first by the hemolymph node, later by the ordinary lymphatic glands.

(5) "The hemolytic function of the hemolymph nodes and hyperplastic lymph glands exceeds that of the primitive spleen, causing an excessive destruction of red cells. The resulting anemia is later compensated for by an increased activity on the part of the bone marrow. It would appear, therefore, that the removal of the spleen leads to an increased production or retention of some hemolytic agent usually disposed of by the spleen. The effect of this hemolytic agent is either to stimulate the phagocytes in the hemolymph nodes to increased activity, or to so change the red cells that they are more easily destroyed by these phagocytes."

There are several additional conclusions which are irrelevant, but the point of interest centers in the fact that in the present case there exist a large number of lymph nodes resembling those which Warthin describes as a primitive embryonal type of the more fully developed lymph node, and that these nodes contain in their substance large bundles of fatty tissue suggesting their "formation out of lobules of fat tissue."

* That the statement used by Warthin in this conclusion, "A new formation of hemolymph nodes out of lobules of fat tissue," may be fully understood, I quote the following from the body of the article: "The first stage in the development of a lymph node from adipose tissue is a dilatation of the small capillaries lying between the fat cells and an infiltration of lymphocytes along their walls. If, now, the capillaries become angiectatic and converted into blood sinuses through the formation of a reticulum arising from endothelial proliferation, the lymph node is formed; if, on the other hand, the capillaries remain small or undeveloped and the lymphoid tissue increased at their expense, while the lymph vessels became enlarged and changed into sinuses, the structure is that of a lymphatic gland. The essential difference between the two is the degree of development of either blood capillary or lymph vessel into blood or lymph sinus."

The case further shows marked hyperplasia of the pre-existing lymphatic tissues and evidence of increased activity of the bone marrow.

The more minute histological studies of the apparent hemolymph nodes in this case have not been made, inasmuch as Warthin's paper came to my notice too late to make this possible. One, therefore, cannot say whether or not they show all the changes manifested in those studied by Warthin in his experimental cases.

I have been content to call this a case of enlargement of the spleen with anemia. I do not feel that we have arrived at any justifiable classification of these cases of enlargement of the spleen associated with anemia and sometimes with cirrhosis of the liver. I am inclined to favor the view that these lesions are not the primary factors, but the results of some previously existing and as yet unrecognized condition. In some respects this case is unlike any of those I have found recorded in the literature. Clinically, it has many analogues. Pathologically, the spleen shows a rather greater increase in the fibrous elements and more marked destruction and atrophy of the Malpighian bodies. The peculiar tendency to general proliferation of the fibrous tissues and the remarkable fat deposits I have not seen recorded.

The etiology of this case is obscure. It is always difficult to exclude the possibility of syphilis. It has been impossible to obtain information regarding the father. No evidence of syphilis could be found in the mother, and there certainly seem to be no very suggestive evidences of syphilis in the patient. There was no history of retarded development nor, in fact, of anything to suggest serious nutritional defects in early life.

Bacteriological examinations were not made, save that sections of the spleen were stained for microorganisms with negative results, but it is not likely, in the light of the results of such studies in similar cases, that they would have thrown any additional light upon the origin of this case.

When this patient first came under my notice, he received inunctions of mercury for some time without any evident effect. Splenectomy was suggested to the family, but declined. The difficulty experienced in removing the spleen, post mortem, suggests the probable wisdom of this decision. Why the spleen is not removed more commonly in these cases of chronic splenic en-

largement, especially in those in which frequently recurring hemorrhages are constantly threatening life, I do not quite understand. Even in the cases in which hemorrhages do not occur, the relief which would follow the removal of this mechanical obstruction to the functions of so many of the organs of the body would probably be complete and permanent. The ability of the lymphatic structures to take on the functions of the spleen has been clearly demonstrated, and in these long-standing cases this property of the lymph nodes is so far developed that its removal probably would not cause even temporary disturbance of function.

I have not given any special attention to the percentage mortality of the cases which have been operated upon, but my impression is that it is not sufficiently high to make the operation inadvisable.

DISCUSSION.

DR. WENTWORTH.—These cases of chronic enlargement of the spleen, associated with more or less anemia, should not be classified as a disease of the spleen. The term "splenic anemia," which has been applied to these cases, implies that the anemia is secondary to a primary disease of the spleen; this assumption lacks proof. The histologic changes in the spleen are those of chronic hyperplasia. This condition is a common one, and is due to a number of causes. There is a more or less marked increase in the connective tissue in the spleen affecting the pulp, the follicles and the blood-vessels. Bacteriologic examinations of the splenic tissue have given negative results, as have also inoculation experiments. The blood changes are those of secondary anemia; in some cases severe, in other cases very slight. With such negative evidence as this, we are not justified in giving a specific name to a condition which is so often associated with chronic diseases in other organs and tissues.

DR. MARTIN.—It seems to me that we have a great many cases of enlarged spleen without any known cause. I have seen a number of cases where the spleen was enlarged and yet the blood changes were not such as to justify the name anemias.

DR. CHRISTOPHER.—I hope at the next meeting to discuss the question from a clinical standpoint. There is no doubt that an infectious element is present, but I am convinced that it is of sec-

ondary importance and that autotoxemia is the cause. There is general emaciation, always associated with abnormal urine, general acidity extremely high and failure on the part of the bowels to perform their normal function. I consider that this type of case presents as distinct an entity as anything I know of. I find that I get better results when I take the cases away from the breast and that milk is the most toxic food for them.

DR. HAMILL.—The anemia in this case was of a mild degree at all times, and was only emphasized on account of existing leucopenia. I agree with what Dr. Wentworth has said in regard to our lack of knowledge of the etiology of these cases, but I am at variance with him in believing that this particular type of case is of common occurrence. I have seen many instances of enlargement of the spleen in infants and children. The condition is exceedingly common, but I have never seen any, excepting this case, in which the enlargement was so great, the course so chronic, and in which hemorrhages occurred. I do not believe, in short, that these cases are in any way related to the more common forms of splenic enlargement with which Dr. Wentworth has placed them.

SOME REMOTE DISEASES ARISING FROM TONSILAR INFECTION.

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That the tonsil may be the primary seat of infection, producing lesions in remote parts, has been known for some time.

The paradigm is diphtheria, which has always been accepted by a certain number of authors as primarily a local infection, a fact that has been verified as the result of bacteriological research.

Erysipelas, in some instances (Gerhardt) has been mentioned, and undoubtedly does occur as primary tonsilar erysipelas.

Baumgarten referred to the tonsils as a possible entrance place of tuberculosis, a fact that has been fully verified by Friedman, the latter author claiming that in young children, especially, it is the seat of primary infection due to food infection.

Leyden, Meyer and Singer have shown that the tonsil is first infected in articular rheumatism; and Menzer has demonstrated the presence of streptococci in the peritonsilar tissue in this disease. Menzer has also shown that the same organism is found in the tonsils that is found in the tissue excised from a case of erythema nodosum. He expresses the belief that in a large number of infectious diseases—pneumonia, scarlatina, measles—the primary infection takes place in the tonsils.

For scarlatina this has been surmised for some time. Dowson, in 1893 stated that the essential lesion of scarlatina is to be found in the throat, just as a chancre is the primary lesion of syphilis. Further than this we are unwilling to follow Menzer. Packard, in an excellent paper on endocarditis occurring in the course of tonsilitis, gives the whole literature on this subject, and reports 5 cases of his own. The connection between angina tonsilarum and endocarditis, I am sure, will be verified by all who have studied their cases carefully. Severe infections of the tonsil have been held accountable for cases of septico-pyemia.

The object of this paper is to add two more remote pathological conditions due to infections of the tonsils to those mentioned.

The mechanism of the production of general infection by local infection of the tonsils is a matter still somewhat unsettled.

The tonsil represents part of almost the first barrier to infection in the mouth; all the structures of this barrier, including that of the tonsil, are essentially a mass of lymphoid tissue. On account of this structure of the tonsil we have been in the habit of ascribing to it all those functions which belong to bodies of similar structure, like the solitary follicles of the intestines, for instance, especially in connection with absorption. Unfortunately, this assumption has been difficult of verification; from investigations that have been made (Hodenpyl) it is still doubtful whether, with undisturbed epithelial covering, substances will be absorbed by the tonsil at all. Stoehr's observations that leukocytes wander through the epithelial layer to and fro, while freely accepted, do not seem to help us very much as far as absorption of bacteria is concerned; in that it is supposed that the leukocytes act only as phagocytes. Menzer has shown that in 3 cases of articular rheumatism streptococci were found in the peritonsilar tissue, especially within the blood-vessels, and believes this to be the method of general infection in this disease, as well as in endocarditis. It must be noted that Singer has found the streptococcus within the lymphoid tissue of the tonsil. But having such indubitable evidence before us as that of Menzer, we must be willing to admit that one method of absorption must be by the route of the blood-vessels of the peritonsilar tissue. The question that arises is whether this is the only route. Certain it is that bacteria have not been frequently found in the tonsils, at least not as frequently as our clinical evidences would warrant us in concluding they should be found. There are various reasons for this: It is possible to conceive of the lymph currents being so rapid that bacteria are carried through the tonsil into the general lymphatic circulation without multiplying within the tonsil; the large number of phagocytic cells may make it impossible for bacteria to develop; and, lastly, the observers who have studied this question may have used methods or material not adequate for a solution of the question. In regard to this latter, it would seem that in some conditions we are justified in suspecting these methods; compare the statements made by older observers in regard to the frequency of the tubercle bacillus in the tonsil with those of Friedman. (Hodenpyl found tubercle bacilli only once in nearly 200 cases; Fried-

man in 24 out of 91 postmortem examinations, but only once in 54 excised tonsils in the living.) Phagocytosis is more or less a doubtful quantity in preventing development of bacteria; after all, accepting the phagocytic theory for all that is claimed for it, when the number and virulence of the bacteria exceeds the number and resistance power of the phagocytes, the bacteria will enter. As to the rapidity of the lymph current in the tonsils no observations have been made that would lead us to believe that this becomes so great as to flush the tonsils of bacteria. If bacteria pass through the tonsils we would find them in the next barrier, the lymph nodes at the angle of the jaw. While these lymph nodes receive the lymph from the nose, the pharynx, the root of the tongue, and the tonsils, and while we know that inflammatory conditions of any one of these localities may be followed by enlargement of these lymph nodes, yet almost daily experience has shown us that amygdalitis of any sort is always followed by enlargement of the glands.

So that in view of recent bacteriological investigations, and as a result of clinical evidence, it seems to me proper to conclude that bacteria are taken up by the tonsils. In addition it might be urged that if one bacterium is taken up by the tonsils, others may also enter; this is accepted for diphtheria; it has been accepted for tuberculosis; and pus producers have been found.

The next step in the production of remote infection is of more general importance. We must admit that the bacteria may be carried into the general circulation by one of two ways, either by the lymphatic current, or by the blood. For our present purpose it is immaterial in which way this is done. But it is of the utmost importance to decide whether the bacteria appear in the general circulation, as they might do irrespective of the route. At the present time the view is gaining ground steadily that, in all infections, bacteria are found in the blood; indeed, it is claimed by some, Adami and his pupils, that bacteria are always found in the blood in health. Older statements can hardly be considered as bearing testimony to the correctness of these views, as they all have been made as the result of faulty methods. At the present time the method first described by Sittmann, with proper modification, is the only one that can be relied upon; taking large quantities of blood (as much as 40 cc.) from a vein, with a syringe, and immediately cultivating in large quantities of a fluid

medium (from 300 to 500 cc. of bouillon). The more closely these directions are followed, the more constantly are bacteria found in the circulation. This has been conclusively shown by Prochaska, in pneumonia; by Cole, in typhoid fever, and, especially, in this disease by Courmont who has found the Eberth bacillus in the blood of every typhoid fever patient he has examined.

In malignant endocarditis, which is a septicemia, it has been shown by Lenhartz that lower forms of life are present in 80 per cent. of all the cases. I may add, that all the work that has been done in connection with the examination of the circulating blood in children, as far as the constant presence of bacteria is concerned is unreliable. Most of it has been done by taking a few drops of blood from the ear of the patient, and then inoculating solid media. For the present, until the method is absolutely fixed, in the adult, as to the quantity limit of blood and culture, no definite results can be obtained in children except in the presence of large quantities of bacteria.

If we now have, in addition to the presence of bacteria in the blood, a *locus minoris resistentiæ,* modernly called diminished local resistance, we have the conditions necessary to explain the production of local manifestations from infection of the tonsils by bacteria.

There is one other method by means of which remote effects can be produced as the result of infection, viz., by the production and localization of toxins. Experimentally, this method apparently stands on a sound basis; whether or no the future will continue to verify its existence in the human being is a question that cannot be decided at the present time. The injection of toxic bodies has been followed by lesions in various parts of the body; but we must remember the stand taken with infection by the Fehleisen bacterium, which was at first looked upon as purely local, but now understood as making remote invasions. This seems to be the case with many other lower forms of life. Where at first the remote effects were supposed to be due to the toxin, it has been shown that they are the result of the bacteria themselves.

The first condition following angina tonsilarum as the primary infection is illustrated by the following history:

A boy eleven years of age came under observation on March 20, 1902, complaining of headache. He is of a neurotic family

on the mother's side; nothing of importance on the father's side, except that his father has been successfully operated on for perforating appendicitis. The boy himself has had measles and whooping cough, otherwise has been well. Upon examining him on the afternoon of March 20th I found him with temperature of 103°, pulse in ratio, and a follicular angina.

Culture on Loeffler's serum showed the presence of a mixed infection, staphylococcus albus and streptococcus. The angina ran a normal course, developing on both tonsils simultaneously, so that by March 22d the temperature became normal. On the evening of this day the patient began to complain of vomiting and great pain in the abdomen; temperature, 102°; pulse, 110. Examination showed the patient lying upon the back, in the position peculiar to abdominal inflammations, very quiet except when he uttered suppressed moans on account of his pain; the abdomen was very sensitive; upon pressure sensitiveness was rather sharply localized in the right lower segment. McBurney's point was sensitive upon the slightest pressure. The colon was empty. An ice-bag was ordered; the boy was put on fluid diet when the vomiting should cease. A small dose of opium was ordered if the pain was not relieved by the ice-bag. No cathartics were given; first, because calomel had been given the day before the onset of the appendicitis; second, because the colon was empty; and, thirdly, because there were no general indications for their administration. On the second day of the illness the vomiting had ceased; the temperature was 102° F. in the morning, the pulse 100, the local symptoms very severe, a slight resistant mass being felt in the right lower abdominal segment. On the evening of this day the temperature had fallen to 100° F., and the pulse to 90; the patient resting more easily, and passing a good night.

The morning of the third day found him without fever; pulse still 90; pain abated, except upon motion; but the resistant mass somewhat increased. He was now ordered lavage of the intestines, which seemed to give some relief; this was continued daily until complete recovery took place. The patient remained in the condition described for the third day, the pain and sensitiveness gradually diminishing (including McBurney's point), and the tumefaction gradually disappearing, for eight days, when he was allowed to get up and go out. On the fourth day of the appendicitis there appeared an erythema upon the abdomen, which devel-

oped successively upon the thighs, the arms, and the back. During the course of the disease there were no manifestations on the part of any other organs. In brief, then, we have a characteristic case of appendicitis, developing in about three days after the onset of an amygdalitis. The explanation for this fact, I believe to be not very difficult to find: the appendix is a veritable *locus minoris resistentiæ,* on account of its being a rudimentary organ as to its structure and its functional activity. It has been repeatedly shown that when bacteria are injected into a lower animal they develop in such places in which the tissues have been previously affected in one way or the other; thus, Pawlowsky has shown that when trauma is produced in an animal, and bacteria are then injected into this animal, the injected organisms are localized at the place where the injury was produced.

At present it does not seem wise to lay too much stress upon the similarity of structure between the tonsils and the appendix; it may be hereafter shown that certain lower forms of life have certain predilections for certain structures; from a clinical point of view this is very attractive, but, as yet, has not been demonstrated. In regard to the presence of lower forms of life in the blood, I have no evidence to offer, for the reasons expressed before; but in appendicitis I have seen the evidences of general infection so often that I am sure these organisms can be found in the blood. The last case of this sort that has come under my observation, which will be reported in detail at some other time, is one in which an operation was done for appendicitis, no rupture, but the appendix containing staphylococcus albus pus; three days after the operation the patient had uremia from acute nephritis; with this an endocarditis; after two weeks a pyelitis. This patient had also suffered from sore throat; but disregarding this fact, it would be impossible to conceive of the conditions described above as existing without the presence of bacteria in the blood, and, considering the short time that had elapsed between the operation and the breaking out of septicemic symptoms, in all probability the infection did not come from the appendix.

In angina tonsilarum followed by appendicitis, the mechanism is the same as is shown by Lenhartz to exist in ulcerative endocarditis; blood infection and infective conditions upon the valves of the heart; in appendicitis we have the same condition, blood

infection from the tonsils, infective conditions in the appendix,* and sometimes marked evidences of general infection.

The second class of cases I wish to report are those in which jaundice followed an infection of the tonsils; of these there are 5 cases, 3 in one family, all occurring in the winter of 1900-1901. As I had occasion to see quite a number of patients with jaundice during this season, it does not seem to me improper to speak of this form as epidemic jaundice. In all the cases, regardless of the primary origin of the jaundice, the symptoms were approximately the same, so that a detailed description of each case seems unnecessary.

The form of sore throat varied; in all except 2 there was a mixed infection, staphylococcus albus and streptococcus, in 1 a pure streptococcus sore throat; in the other that which is in my experience exceedingly rare, a pure staphylococcus aureus angina. The time elapsing between the angina tonsilarum and the jaundice varied from three days to ten days. The first symptoms noted were pain in the epigastrium and vomiting, a moderate amount of fever (maximum, 101° F.); this continued for four days on an average.

After two days slight icterus; bile coloring matter in the urine; clay colored stools; itching of the skin; slight drowsiness and enormous appetite. In all of the cases the region of the liver was sensitive to pressure, the gall bladder not full, and the spleen was enlarged in 3 cases. The jaundice lasted, depending upon the intensity of the disease, from three to five weeks, and in all cases there was, at some time, subnormal temperature which did not disappear until the patient had fully recovered. The three patients in one family, age five, seven and a half and nine years, all had the same infection of the tonsils, but the infection evidently had grown less virulent as it passed through the second and third child, in that the attacks of jaundice in the latter were much less intense. Albumin was not found in the urine of any of these cases. No pathogenic organisms were found in cultures made from the stools. There was no mortality. In regard to the acceptance of the term "epidemic jaundice" there can be no discussion, in that the occurrence of epidemics of jaundice has been reported by a number of authors. (Kissel in his excellent article

* The relation between appendicitis has already been referred to in Packard's paper. In Germany also this subject has received attention.

mentions twenty-one authors, German, French, English and Italian.) It is, however, strictly proper to hold differing views upon the existence of that form of disease called infectious jaundice, notably in regard to its frequency. This form of jaundice has been called Weil's disease, for the reason that this author (1886) found a combination of symptoms, jaundice, nephritis, enlargement of the spleen, with fever, which seemed to justify him in describing what he conceived to be a disease *sui generis,* and not related to the ordinary forms of jaundice. Since that time it has been repeatedly shown that (*vide* Kissel) variations occur in all directions as far as development of symptoms is concerned; indeed, Hennig claims that of Weil's combination of symptoms the most important one, jaundice, may not exist, and yet the same clinical conditions may be present. Again, cases occur running their course so mildly or so severely that Weil's description does not hold good. Finally, the French authors (especially Chauffard, 1885) have described the same conditions so thoroughly that all forms were considered before Weil's paper appeared; therefore, no reason exists for either calling this form of disease Weil's disease, or of taking it out of the class of the ordinary forms of jaundice. As to the mechanism of the production of this form of jaundice we are by no means well informed. It has been clearly shown that jaundice may be produced as the result of the introduction into the circulation of certain toxic bodies; phosphorus (Wyss, Ebstein) and santonin (Cramer). It has not been shown experimentally that the bacterial toxins produce jaundice.

For bacterial causation we find more evidence: Luerman and Jehn, describe epidemics apparently due to vaccination; of 96 cases reported by Kissel, 14 had some catarrhal affection of the respiratory tract; he states that a number of authors have seen jaundice in connection with influenza, a fact to which I can also testify; jaundice occurs in typhoid fever, the Eberth bacillus being found in the gall bladder as well as in the large biliary ducts. It also occurs in cholera and other acute infectious diseases. The finding of the typhoid fever bacillus in a gall bladder of patients without typhoid fever lesions in the intestine is of great importance in showing that bacilli which are found in the biliary passages must not, of necessity, ascend from the intestine but may come from the blood. As a matter of fact,

experimental evidence shows that when bacteria are injected into the blood they are eliminated by the liver and the kidneys, a fact again verified by Pawlowsky. Whether these lower forms of life produce jaundice either by causing an obstruction of the interlobular bile ducts, by producing a cholecystitis, or in any other way, is a matter of secondary importance, in view of the fact that it has been shown that they may produce jaundice. As far as the bacteriology of infectious jaundice is concerned, this is still in its infancy, but we are justified in the conclusion that bacteria being present in the blood, from any local infection, they leave the body by way of the liver, and jaundice may follow.

There is one other method by means of which jaundice could be produced from a remote infection, viz., by way of the gastro-intestinal tract; a gastritis being produced which is followed by a duodenitis. At the present time there seems to be a tendency to minimize the possibility of production of jaundice in this manner. However this may be, in our cases this manner of production of jaundice may be excluded; there were no evidences of gastritis except the vomiting, and this was largely reflex, coming on with the pain and disappearing when this grew better; but, above all, the gall bladder was not full. When there is obstruction in the ductus communis choledochus sufficiently well pronounced to prevent the greater part of the bile from flowing into the intestine, the gall bladder is always distended. In children the gall bladder is more easily palpated than in adults, and yet in not one of these patients was the gall bladder found distended.

DISCUSSION.

DR. JACOBI.—Do you think it is necessary for the epithelial covering of the tonsils to be interrupted for the admission of infection? I believe that is a mistake. There are very few normal tonsils, particularly in adults. There are changes in the tonsils as a result of the frequent diseases of infancy. But, even normally, the epithelium of the tonsils is not contiguous, as Stoehr proved thirty years ago.

DR. FREEMAN.—A case somewhat corresponding to Dr. Forchheimer's first case came under my care. The patient was a seven-year-old boy with sore throat, and tonsils which were somewhat enlarged and very red; slight fever and pain over his appendix, and with rising temperature. On the third day there appeared a

slight blowing murmur over the apex of the heart. Salicylate of soda was administered, the temperature went down and the symptoms disappeared.

DR. CHRISTOPHER.—About four years ago I noticed a number of cases of what were clinically bronchitis, terminating with crises, and where there was no lung consolidation. I attempted to look into the epidemic which was prevailing and examined 200 cases, made cultures from every case of so called grippe that was met with, and in only two or three of these cases were there any influenza bacilli found, but there were pneumococci, staphylococci and streptococci. I learned to look upon certain throats as abnormal which I had previously regarded as normal. The enanthem was not limited to the tonsils at all, but the characteristic feature was its manifestation upon the soft palate. The free edges of the anterior pillars were found reddened and extending upward on both anterior pillars, having a well-marked line of demarcation and involving the uvula. With this there was always tonsilar involvement, which was more difficult to make out than the redness in mild cases, and it is less distinctive. A number of these cases showed cultures of pneumococcus. In addition there was occasionally exudate of the pultaceous variety. The location of this upon the soft palate is, to my mind, as diagnostic as anything could possibly be from a clinical standpoint; and I would rather make a diagnosis of pneumococcic sore throat than diphtheria from a macroscopic examination. Involvement of many organs occurs in these cases. One case had earache, and three days later, when the ear was opened, pneumococci were found in the pus. On the fifth or sixth day the lung consolidated, and this consolidation continued without any tendency to resolution, and the child died. Endocarditis and nephritis developed and a terminal meningitis. In a great many of these cases endocarditis has been found. It is a form of endocarditis that disappears, but returns again every time the child gets a cold.

In addition I have found one case where the appendix became involved. The case was an absolutely typical one with a typical throat as described. I first saw the boy in the evening, and the next day he complained of pain in the side and said: "Doctor, I have appendicitis." He said he had had it two years before, and knew that he had it again, and it was so. The case was seen by two surgeons who declined to operate. One of the surgeons said that it was a quite usual experience to him to find appendicitis in grippe, and that the cases always recovered without operation. The headache and backache disappeared with the changes in the appendix. One peculiar manifestation I have found in connection with this is enlarged lymph nodes in the neck, with sensation of suppuration. In one particular case, where we had the pneumococcus present, the nodes seemed to me so certainly to contain pus that I had Dr. Fenger see it, and he agreed that they probably

contained pus, but would like to postpone opening them for a day. The day following, the temperature dropped. It was as sharp a crisis as I have ever seen, and within a few hours the swelling of the lymph nodes disappeared. In a few weeks another case of the same kind came under my care. In neither of these cases was there involvement of the lungs.

DR. JENNINGS.—During the past year I saw in Detroit an epidemic similar to that just described by Dr. Christopher, and I made bacteriologic studies with the same experience that he has related. The cases were those of upper respiratory pneumococcus infection. I have not noticed the peculiar pharyngeal phenomena described by the doctor, but the pneumococcus infection is pretty certainly the cause of these epidemics of pharyngeal catarrh.

DR. FORCHHEIMER.—I agree with Dr. Jacobi as to the contiguity of the epithelium of the tonsils, but I think that our clinical evidence that bacteria are taken up by the tonsils is too strong not to be believed. The tonsil does have bacteria within it frequently, not only tubercle bacilli, but other forms. I am glad to have heard the confirmatory reports in regard to the connection between jaundice and appendicitis and the localized infection. That was the point I wanted to bring out. I have seen a number of cases where jaundice followed infection of the bronchial tree. The great point is to recognize that in the majority of these cases of infectious disease we are dealing with a septicemia and as we go further a great number of diseases will be added to this list. I want again to call attention to the tonsil as a place of localized infection in these septicemias. I do not think all these infectious diseases begin in the tonsil, but I am sure that a large number of the general infections arise from the tonsil, and that this fact has been overlooked.

CONCLUSIONS AS TO THE TREATMENT OF TUBERCULOUS PERITONITIS IN CHILDREN.

FROM A STUDY OF CASES TREATED AT THE CHILDREN'S HOSPITAL FROM 1884 TO 1902.

BY THOMAS MORGAN ROTCH, M.D.,
Boston.

I am at present preparing a report of the cases of tuberculous peritonitis which have occurred at the Children's Hospital during the past eight years. This paper will be published later, and what I wish to do to-day is simply to state when, and in what class of cases, laparotomy should be performed with a reasonable chance for recovery, and to present some cases which recovered after the operation for laparotomy.

The total number of cases admitted to the Children's Hospital in which the diagnosis seemed reasonably certain was 69. Of these, 39 were boys and 30 were girls.

In regard to the age, the chief points to be noticed are, *First*, that tuberculous peritonitis in early life is rare in the first year, and when it occurs at this age, it is almost universally fatal, as it is almost invariably part of a general tuberculosis, and is for that reason not amenable to treatment by laparotomy.

Second, that the most common age for the disease to appear is from one and one-half to four years.

Third, that the cases of tuberculous peritonitis may be divided pathologically, into primary and secondary cases. The secondary cases are most frequently those which are infected from the lungs, from the intestines, and from the mesenteric lymph nodes.

Fourth, where the lung or any important organ is the primary cause, the prognosis is that of general tuberculosis, and laparotomy in these cases is seldom of any avail. This class of cases includes those in which there are tuberculous ulcers of the intestine. Tuberculosis of the mesenteric lymph nodes, however, as a primary cause, does not mean that the cases are necessarily fatal, or that they cannot, under certain circumstances, be relieved by laparotomy; therefore, where the tuberculosis is primarily in the

mesenteric lymph nodes, laparotomy is indicated in the treatment, as we have no means of determining in these cases, excepting by trial, whether the laparotomy will be successful or not. Finally, where the tuberculosis is primarily in the peritoneum, laparotomy is essentially indicated and is often followed by complete cure.

Fifth, there is a distinction to be made between those primary cases in which, first, there are many thick adhesions and advanced tuberculous lesions not accompanied by the presence of much fluid; and second, where the tuberculosis is represented by miliary tubercles of the peritoneum with ascites.

In the first class of cases, although the prognosis is not so good as in the second, still laparotomy should be performed, as in a number of these cases complete recovery took place just as it has so often taken place in the second class with ascites. The second class is the most favorable for recovery by operation in children, of all the varieties of tuberculous peritonitis, and laparotomy should be performed without question in this class of cases.

In the series of 69 cases which I have just spoken of as studying at the Children's Hospital, the fatal cases were those in which the tuberculous peritonitis was secondary to a primary tuberculosis of some other important organ, but in a certain proportion of these cases there were only to be found marked local lesions.

The cases of recovery consisted mostly of those in which there were no advanced tuberculous lesions of the peritoneum, and where there was a considerable amount of fluid in the abdomen. There were, however, some cases where thick adhesions and advanced tuberculous lesions were present in which recovery also took place.

Although it is not yet determined how many years it is necessary to wait before we can say that complete recovery from the tuberculosis has taken place after laparotomy has been performed, yet, judging from this series of cases, there is no doubt that the patient should be given the benefit of the chance; and that laparotomy should be performed, notwithstanding the fact that there are well-known cases of spontaneous recovery. I have arrived at this conclusion not only from a study of my own cases, but also of large numbers of those reported by other observers, and I be-

lieve that this conclusion is correct, because, although a case may get well without laparotomy, the large majority do not, and when we have made a diagnosis of a localized tuberculous peritonitis, we are unable to say whether such localized process may not at any time become general and result fatally.

Taking into consideration also that an exploratory laparotomy, when properly performed, is a safe procedure in comparison to nonoperative treatment, and also considering that merely an exploratory laparotomy in many cases seemingly cures the disease, it is better to operate at once and to allow the patient the benefit of the chance.

The cases which I present for your inspection to-day are only six out of the twenty which were discharged from the hospital relieved. The others have been heard from by letter, but I was only able to get this small number to come to the meeting, as they almost all lived at a distance from Boston.

CASE I.—M. B., girl, age two years and three months, operated on in October, 1897; the operation for the tuberculous peritonitis being complicated by an ovarian cyst which was removed at that time. There was much abdominal distention, a large amount of fluid and no adhesions. She recovered rapidly and has been perfectly well ever since.

CASE II.—H. N., age three years and ten months, operated on May, 1900. Much abdominal distention with considerable fluid, adhesions moderate. Made a good recovery and has been well ever since.

These two cases represent the more favorable class of cases for operation.

CASE III.—F. S., boy, age two years and ten months, operated on June, 1900. There was some abdominal distention with gas, no fluid was found, and there were moderate adhesions. Recovered, and has been well ever since.

CASE IV.—C. L., boy, age one year and eight months, operated on July, 1900. Considerable distention from gas, no fluid, no adhesions. Recovered, and has been well ever since.

CASE V.—B. A., boy, age three years. Some distention from gas, no fluid, moderate adhesions. Made a good recovery and has been well ever since.

CASE VI.—E. P., boy, age two years, operated on January,

1901. Abdominal distention from gas, no fluid, no adhesions. Recovered, and has been well ever since.

Before closing this paper I wish to acknowledge the courtesy of my colleagues, both medical and surgical, at the Children's Hospital for allowing me to report the cases which happened to occur in their respective services, and also to thank Dr. Maynard Ladd for systematizing and arranging the cases from a study of the records.

DISCUSSION.

DR. ADAMS.—This subject opens up a wide field for discussion. As for my colleague in Washington and myself, we have been in favor of exploratory operation ever since these cases first became of interest to the profession. I do not believe they will get well when let alone, and I am in favor of operation at once. We have had three cases of operation and two successes, which I think is encouraging.

DR. ACKER.—What was the condition of the lungs and lymph nodes in these cases?

DR. ROTCH.—All the cases were examined before operation, and we did not operate where there was primary trouble in the lungs or lymph nodes. Where there were primary lesions elsewhere of marked character we considered that laparotomy was not indicated.

DR. FORCHHEIMER.—Did any of your female patients have leucorrhea? Vierordt called attention a few years ago to the finding of the tubercle bacillus in leucorrheal discharges, and in two cases we have found the tubercle bacillus in this way. One of them has just been operated upon for localized tuberculous peritonitis. An interesting question is whether the bacilli ascend, or whether the vaginal infection is a result of the peritoneal. I think we should try other methods before subjecting the patient to a laparotomy, although when properly performed the operation is not attended with much danger. I always use other methods first, however. I do not know the philosophy of their use, any more than we know why cutting into the abdominal cavity and letting in the air kills the tubercle bacilli, but we know it does good, and we certainly know that cases get well without laparotomy. If, after a certain lapse of time, they do not recover, then a laparotomy should, of course, be done. It is difficult to tell what cases should be operated on. If you have chronic lobar or lobular pneumonia, such a case should certainly not be touched, for it will

in all probability die anyhow. If, on the other hand, the process is limited to the peritoneal cavity without evidences of activity in other important organs and the patient has been given a fair chance with both local and general treatment, laparotomy should be done. I want, especially, to put myself on record as taking the view that the operation must not be looked upon as a *dernier ressort,* but as an indication to be filled at the proper time in proper cases.

DR. KOPLIK.—The fact there is a difference of opinion on the subject shows that it is a difficult matter to come to a conclusion about. In some statistics published about a year ago by Herschfeld, in which about 50 cases were collected; he showed that after operation the percentage of recoveries was 33 per cent. Recently there was published a statistical series of cases not operated on, and in which the percentage of recoveries was about the same. So that it is one of the most difficult questions to decide as to whether operation benefits these cases or not. In my service at the hospital we have had in the past year 5 cases: 2 of the miliary type, and the others with large masses in the abdomen. We operated on 3 of the 5. One case possibly of the miliary type was left alone. This patient increased in weight under hygienic conditions and good food and certainly seemed much better when he went out than when he came into the hospital. Another case (miliary) was operated on and recovered. In another case with masses in the peritoneum and no lung infection at all, the result was disastrous. The patient died of exhaustion shortly after operation. Another case with masses is now in the ward, and was operated on a few days ago. I think we must judge somewhat by the character of the cases. If the children are getting along well under good hygiene and increasing in weight and general condition, I shoud leave them alone; if a patient is losing ground with no complications elsewhere, with outspoken tuberculous peritonitis, I would give the child the benefit of the last hope by advising operation.

DR. CHAPIN.—We must bear in mind that these cases sometimes open themselves at the umbilicus, a spontaneous opening. I have seen two such cases. We are very much indebted to Dr. Rotch for this presentation. It furnishes valuable data for reference.

DR. CAILLÉ.—This is an important demonstration, particularly in view of the skeptical attitude of many physicians toward timely operation for this disease. Two years ago I reported to this society 11 cases operated upon. Since then I have had 6 more cases, altogether 17 cases, and of these 17, 4 are alive and show absolutely no symptoms, not even a rise of temperature, which

has been taken for months. A number of other cases are alive but not cured. Some have died. In a number of my cases two, three, and five years have elapsed since the operation.

As regards the indication for laparotomy, I believe that laparotomy is indicated as soon as we make the diagnosis; or better still, as soon as we suspect tuberculous peritonitis. Colleagues who claim cures of tuberculous peritonitis by medication and other means should remember that, as a rule, a positive diagnosis can only be made by opening the abdomen. You may suspect tuberculosis, but you are not sure of your diagnosis.

DR. COTTON.—I remember Dr. Caillé's presentation two years ago, and at that time I mentioned that I had a case under my care and was encouraged by the presentation to operate. When I got home I talked to the mother of the boy and convinced her that an operation should be done. Some other matters claimed my attention for a week or so, and when I returned the boy was making fair progress and the operation was postponed, and finally he recovered without operation. Since then he has gone through a case of typhoid fever. The boy was ten years of age. I have had three cases since that. None of them has been operated upon, two have recovered and the third is promising. As regards positive diagnosis, I agree with Dr. Caillé. I do not see how the diagnosis can be made definitely without laparotomy. A dry peritonitis, with a full abdomen and with evident masses in the omentum, seems to me to be a contraindication for operation.

DR. JACOBI.—I have no figures here either of deaths or recoveries, but I think every one of us sees a good many cases, and I have always been in a position of being compelled to give an account of my experiences and results once or twice a year. They are as follows: we meet tuberculosis that has healed in a great many instances; we find the evidences of that in the living, and more especially in the dead, at autopsies. We find healed tuberculosis in the lungs where we are apt to expect it least; on the pleura and in the bones, and on the peritoneum also. So long as the disease is localized, it is apt to heal, provided the patient is placed in good circumstances, sometimes even under the influence of very bad conditions. As soon, however, as it is disseminated, it is apt to go on and prove fatal. Tuberculosis of the peritoneum is generally in the beginning a localized disease, perhaps more so than pleural tuberculosis. We make a diagnosis of tuberculosis of the pleura and follow it up for years before seeing anything in the lung. Cases of persistent tuberculosis are frequently not attended with fever. The acute cases, however, are of a different character and are more progressive and apt to terminate fatally in a short time. So long as the disease is localized, it is

apt to heal, no matter what you do. Many of my cases have gotten well by simply putting them in good surroundings, with good food and cod liver oil. I have seen a number of such cases with local peritonitis get well under this treatment, and I have seen them get well with laparotomy, or with iodin, or with guaiacol, or with arsenic and other things. But, so long as they are not attended by fever, and so long as the disease is localized on the peritoneum, mostly with ascites, I should not think of urging a laparotomy, because I see so many of these cases getting well without it. What should we do when the tuberculosis has become disseminated, as after a year or two when we find tuberculosis of the pleura and finally of the lungs? Well, then none of our treatment will save the patient. Therefore I do not know if I am not prepared to say that laparotomy does not do anything in such cases, or that it has never cured a case that would not have gotten well without it. I am willing to accept the data given by Dr. Rotch, but, at the same time, I have the impression that the number of cases that recover compared with the whole number of cases is about as favorable on one side as the other.

DR. KOPLIK.—I want to say a word in explanation. No one would say positively that miliary tuberculosis existed without opening the peritoneum. On abdominal examination and through the rectal wall, you can exclude the other forms, and thus make a probable diagnosis.

DR. MILLER.—I should like to put on record here a case of tuberculous peritonitis under my care that recovered. The patient, a girl, aged nine, had the strongest clinical evidence of tuberculous peritonitis. She was put to bed, given good food, good hygienic surroundings, and cod liver oil and iodoform were administered by inunction. The ascites disappeared and the tumors gradually melted away, the patient leaving the hospital after six months in good condition. I am of the opinion, though, that an exploratory operation seldom does harm, and should always be undertaken after a fair trial of medicinal means.

DR. SEIBERT.—It has been stated here that tuberculous peritonitis is a local disease. I am not prepared to accept that statement. To my mind, it is nothing more nor less than a local manifestation of systemic tuberculosis.

DR. ROTCH.—My object, of course, in this paper, was to determine when to operate and when not to. Dr. Koplik says it is his last hope. I can only say that I am surprised that he should take this position in the face of the lack of danger in laparotomy and the results we are getting continually in all parts of the world. You may say the same thing about empyema. A number of cases get well spontaneously, but we do not wait—we operate. You can

never know which case is going to get well without operation, and we do know that a large number will die if not operated on. As to Dr. Cotton's remark about conservatism, I do not think it is conservative not to operate. Of course every one should not undertake the operation, but only the skilled surgeon; the point being that it is safe when properly done. In reply to what Dr. Jacobi says of there being equally good results without operation, I do not think statistics will support his view; they certainly do not in this set of cases. Here we have patients dying without operation, 20; after operation, 12. The cases of spontaneous recovery are rather few, and how can you know in the beginning which cases will be the spontaneous ones? We certainly know that there is danger in waiting. After you know that it has ceased to be a localized condition, then there is no use to operate. It seems to me that unless you are doubtful as to its being a comparatively safe operation you are taking a very unsafe position to say you will wait. Why not operate in the very beginning and give the patient the benefit of the chance? I have had patients get well without operation; but that does not make me postpone operation. Trusting to the iodids is, I think, very unsafe in any case; for at any time the disease may become disseminated and the proper time for operating lost.

DR. JACOBI.—I have not said that I recommend the iodids as a cure for tuberculosis, general or local. I only say that I see patients getting well—I might say, if you wish, in spite of the iodids. They have got well no matter what has been done, even after laparotomy. What I meant to state as my opinion is that we cannot prove that laparotomy cures our cases so long as there are so many cases that get well without operation. If I speak of the treatment of tuberculous peritonitis with guaiacol, rest and good food, it is because I know that this treatment will do some good. As a rule, I treat such patients with arsenic also, given for a long time. I seldom give iron in these cases, because they do not bear it well. It is never tolerated as long as there is an increase of temperature. The best way of relieving the heart and stimulating the circulation generally is with cold water; so I recommend rest, proper diet, guaiacol and arsenic, and cold water, externally with vigorous friction. If I could promise that laparotomy would cure, I would laparotomize them once a week.

DR. ROTCH.—I make the rule to operate at once in suitable cases such as I have endeavored to describe.

TWO CASES OF UMBILICAL FISTULÆ, DUE TO TUBERCULOUS PERITONITIS.

BY GEORGE N. ACKER, M.D.,
Washington, D. C.

CASE I.—W. R., aged five years, male, white, came under my care at the Children's Hospital, April 4, 1902.

FAMILY HISTORY.—Father and mother both living, and in good health. One other child, who is healthy. Tuberculous history on maternal side.

PREVIOUS HISTORY.—Labor and birth normal. Never breast-fed—condensed milk given. Has always been considered a healthy child.

PRESENT ILLNESS.—During the summer and fall of 1901 he had ascites. After remaining in bed for two weeks, this disappeared. There then appeared a hard, swollen mass in the region of the umbilicus. There was no marked abdominal pain. Last October he had pertussis, and cough has persisted since. Two weeks ago, during a paroxysm of coughing, a rupture of the abdominal wall through the umbilicus occurred, and a fecal fistula resulted.

Appetite is very poor. Has a severe cough, loose and bronchial in character. No expectoration. Fever at night; no sweats. Desires to sleep constantly. Micturition free.

PRESENT CONDITION.—Extreme emaciation. No deformities. Superficial lymph nodes are swollen. Chest walls are flattened antero-posteriorly. Expansion is diminished. Percussion note is impaired over left apex. Throughout both lungs, and most marked over left apex, are numerous liquid râles, large and small. Frequent loose bronchial cough. Heart sounds are weak. The second pulmonic sound is accentuated. Pulse is rapid and weak, 130 per minute. Moderate leukocytosis present. Hemoglobin, 70 per cent.

Appetite is poor. Desire for water is constant. The feces are passed through a large fistulous opening in umbilicus. The stools are frequent, loose, yellow and digested, with some mucopurulent material present. The abdomen is distended

and tympanitic. Passing through the center of umbilicus there is present a sinus as large as a ten-cent piece, the edges hard and undermined. The tubercle bacillus was found in the pus. The liver is about an inch below the ribs. It is impossible to feel the spleen.

Intellect impaired and dull, cerebration slow. Lies in semi-

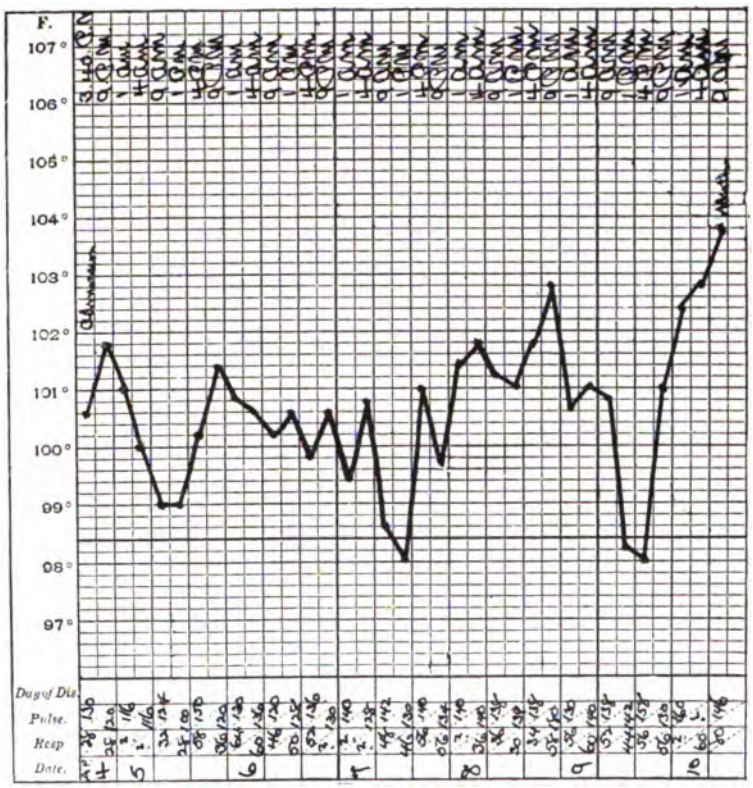

CHART OF CASE I.

stupor; eyes half closed. Sensation and motion unimpaired. Special senses normal. Voids urine involuntarily at times. Urinalysis negative.

PROGRESS OF CASE.—The child had loss of appetite with profuse sweats and died of exhaustion April 10th, after being in the hospital six days. (See Chart of Case I.)

NECROPSY.—The umbilical fistula lead down to a large, walled

off cavity formed by adhesions to the abdominal wall. From this there was a second fistula leading into a large mass of adhesions and enlarged lymph nodes, from which fecal matter exuded. This connected with a rupture in the lower one-third of the ileum at the base of a large tuberculous ulcer—the tear being about one-fourth of an inch in diameter and situated in the lumbar region. There were firm adhesions present, binding and matting viscera and intestinal coils. Tubercles were seen everywhere.

All the mesenteric lymph nodes were markedly enlarged and caseated. Numerous ulcerations were found along the course of the small intestines. The spleen was infiltrated with tubercles. The liver was somewhat larger than normal, with few caseating areas. The kidneys were normal.

The lungs were universally involved. The left apex was consolidated. The bronchial lymph nodes were swollen and softened in places. The heart muscle was pale and soft. There was marked congestion of the brain, but no tubercles. The tubercle bacillus was demonstrated in the various organs and glands.

CASE II.—M. F., aged eight years, female, colored, entered Children's Hospital April 27, 1902.

FAMILY HISTORY.—Father and mother both living and in good health. Patient is the only child. Tuberculous history on paternal side.

PREVIOUS HISTORY.—Labor and birth were normal. Breast-fed. Dentition normal. About four years ago had severe attack of adenitis. Lymph nodes of neck and axilla were involved. Cervical lymph nodes were excised and a sinus has persisted since. Has had pertussis. Subject to attacks of acute coryza.

PRESENT ILLNESS.—Six weeks ago was taken with severe pains in abdomen, followed by ascites, diarrhea and progressive emaciation. Seven days ago rupture of abdominal wall occurred through umbilicus, with mucopurulent discharge tinged with fecal matter. Bowels are constipated. Has a severe cough.

PRESENT CONDITION.—Body very poorly nourished; emaciated; no deformities. Face is puffy and swollen. Expression is dull and heavy. Apathetic, except when disturbed, then she becomes restless and excitable. A few cicatrices are present on neck and upper portion of thorax. Three fistulous openings are present, one on either side of neck and one in upper and anterior portion of right axilla. These discharge a small amount of seropuru-

lent material. The superficial lymph nodes are enlarged, especially in the cervical region. There is slight nasal discharge.

She has a frequent loose bronchial cough. Respiratory move-

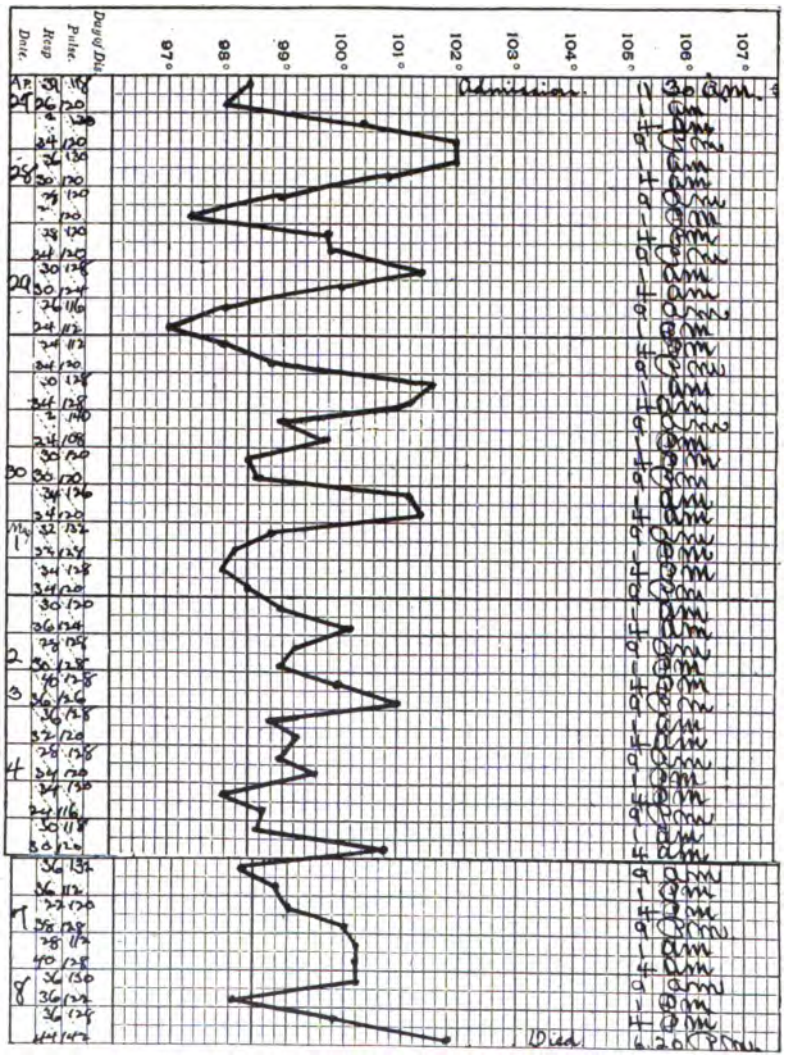

CHART OF CASE II.

ments are restricted on right side. Dullness on percussion, with harsh breathing, and large mucous râles are present over right apex. Moist râles, both large and small, are found throughout

both lungs, less numerous on left side. Heart sounds are good. Peripheral circulation is not good; extremities are cold and nails get blue. Leukocytosis present. Hemoglobin, 58 per cent. Digestion is good. Appetite is ravenous. From one to four loose yellowish stools in twenty-four hours. Abdomen is markedly distended and tympanitic. Liver and spleen are not palpable.

In the centre of the umbilicus is a small opening about the size of a pin's head, clear cut and sharply defined, surrounded by an area of redness and infiltration. From this opening exudes a small amount of yellowish serum, having a fecal odor.

Intellectual faculties impaired. Mind wanders at times. Voids urine involuntarily at times. Sp. gr., 1011. Reaction acid. Albumin, slight trace. Urea, gr. v to the f℥j. Microscopical examination negative.

The diazo reaction was negative. Three tests were made.

PROGRESS OF THE CASE.—There was very little discharge from the fistula until the 29th of April, when a second fistula appeared to the left and half an inch below the first one. The child became weaker, and was in a semi-stupor nearly all the time, and died on May 8th. (See Chart of Case II.)

NECROPSY.—Only partial examination permitted. The fistula was traced downward and to the right, leading into a sac walled off by fibrous adhesions. From this a second opening was found, entering a large mass of adhesions and enlarged mesenteric lymph nodes. There was in communication with this a rupture of the middle portion of the small intestine, situated just above the bladder and discharging liquid feces. Adhesions were everywhere present, the omentum was thickened and all parts studded with miliary tubercles. The spleen contained small yellowish masses. The bronchial lymph nodes were enlarged. The apex of the right lung was consolidated.

These cases I think, illustrate very well the condition described as periumbilical tuberculous abscess. Both of them ran a very rapid course. Recovery has been reported in such cases even after a fistula has discharged pus for months. In both of my cases the tuberculous process commenced in the thorax, probably in the bronchial lymph nodes, and afterwards involved the intestines, from which it passed outward to the peritoneum. The rupture at the umbilicus was due to a weakness at this point owing, no doubt, to a congenital defect.

TYPHOIDAL APPENDICITIS IN CHILDREN.

BY A. SEIBERT, M.D.,

New York.

The only excuse for relating the histories of the following two cases is to call attention to a complication of typhoid fever in children which, on account of its rarity, has been somewhat neglected by pediatric writers. While Osler ("Practice of Medicine," p. 24), when speaking of typhoid in adults, states that "perforation of the appendix is not very uncommon," neither Rotch nor Holt mention appendicitis as a possible complication of typhoid in their pediatric text-books. Of the foreign authors Baginsky (Berlin), Ashby (Manchester) and Filatow (Moskow), also omit this subject in their text-books.

In my personal experience I do not remember ever having recognized typhoidal appendicitis in a child before, but am inclined to think that this was not due to its entire absence, but simply to an oversight on my part. In fact, I see no reason why typhoidal appendicitis should be less frequent in children than in adults, as both affections are common enough during the first ten years of life.

CASE I.—Abraham M., aged eleven years, had been suffering from headache, somnolence and languor for over a week, but had attended school, when suddenly, on May 19, 1901, he was seized with sharp pains in the abdomen. The family physician found the boy very ill, with a rectal temperature of 106.2° F., and what appeared to be stomach cramps. Enemata and calomel reduced the temperature to 105° F. the next day, but the pain remained the same. I saw the boy in consultation during the ensuing evening and observed the following: temperature, 104.6°; pulse, 96; occasional sighing; apathy; slight muscular tremor; slow speech; spleen slightly enlarged, no tympanites, but marked tenderness over the right abdomen, slight dullness over the right iliac fossa.

In attempting to diagnosticate, it was at once clear that the high temperature and comparatively slow pulse, the somnolence

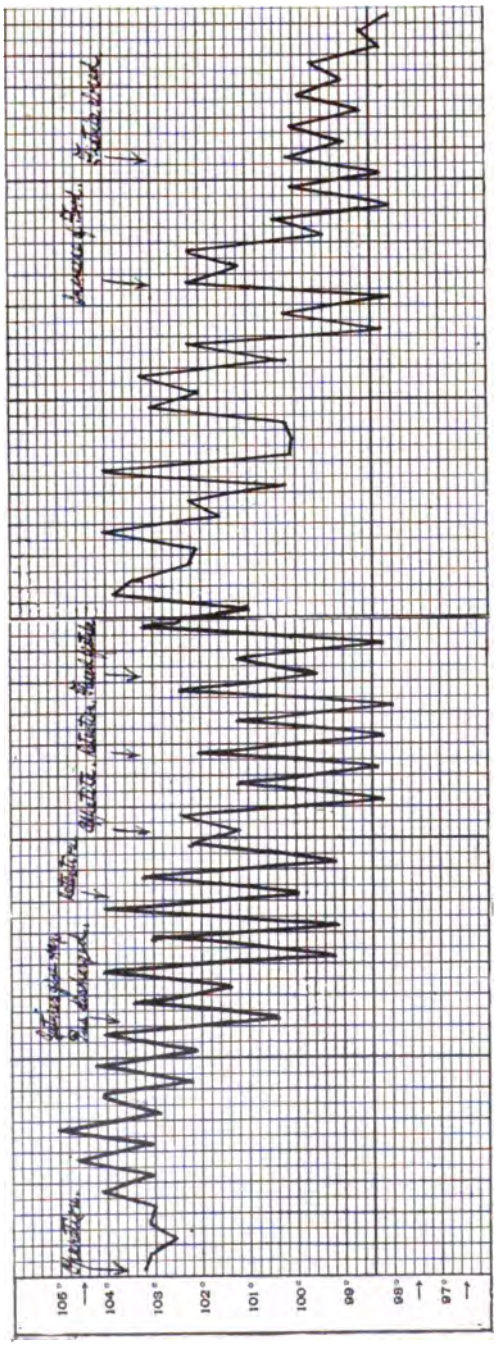

CHART OF CASE I.

and the headache, pointed to typhoid infection. As to whether the local peritonitis was due to perforation of a typhoid ulcer or to appendicitis, was left an open question, to be answered after laparotomy (which I urgently advised) had been performed. The patient accordingly was removed to St. Francis's Hospital, where Dr. F. Kammerer removed the ulcerated appendix the next morning. The visible intestine was found to contain numerous typhoidal infiltrations. Widal's test gave a positive reaction.

The further history of this case (as illustrated by Chart no. I.) is but of interest in so far as the high temperature and the other symptoms of typhoid disappeared in the characteristic manner within two weeks under fluid diet without milk; that the wound discharged pus freely for nearly five weeks after the operation, with frequent retention and septic fever; and at last by the formation of a fecal fistula three weeks after the operation. This, together with the wound, had closed at the end of the seventh week. The boy's appetite had returned two weeks after the operation.

Rectal irrigations, otherwise always used by me in typhoid, were not employed in this case, for obvious reasons.

Judging by the history and the clinical symptoms we may safely surmise that the infection of the appendix occurred during the second week of the typhoid fever.

CASE II.—Albert B., aged ten years, was admitted to St. Francis's Hospital on August 24, 1901. His illness began two days before, with headache, diarrhea, epistaxis and cramp-like pains in the abdomen. The pains remained constant in the right iliac fossa. Epistaxis occurred on two occasions. The diagnosis of appendicitis was made on admission. The patient was operated upon on the same day. A congested and thickened appendix with adhesions was removed. Primary union of the wound resulted.

Five days after the operation the temperature (see Chart no. II.), which had not come down to the normal, slowly began to rise again. During the fourth week I was requested to see the patient in the surgical ward and found evident symptoms of typhoid fever. The Widal test gave positive reaction. Calomel on the first day, then two regular rectal irrigations with three pints of luke-warm water (made without a tube), fluid diet, without milk, and hydrochloric acid before each meal materially

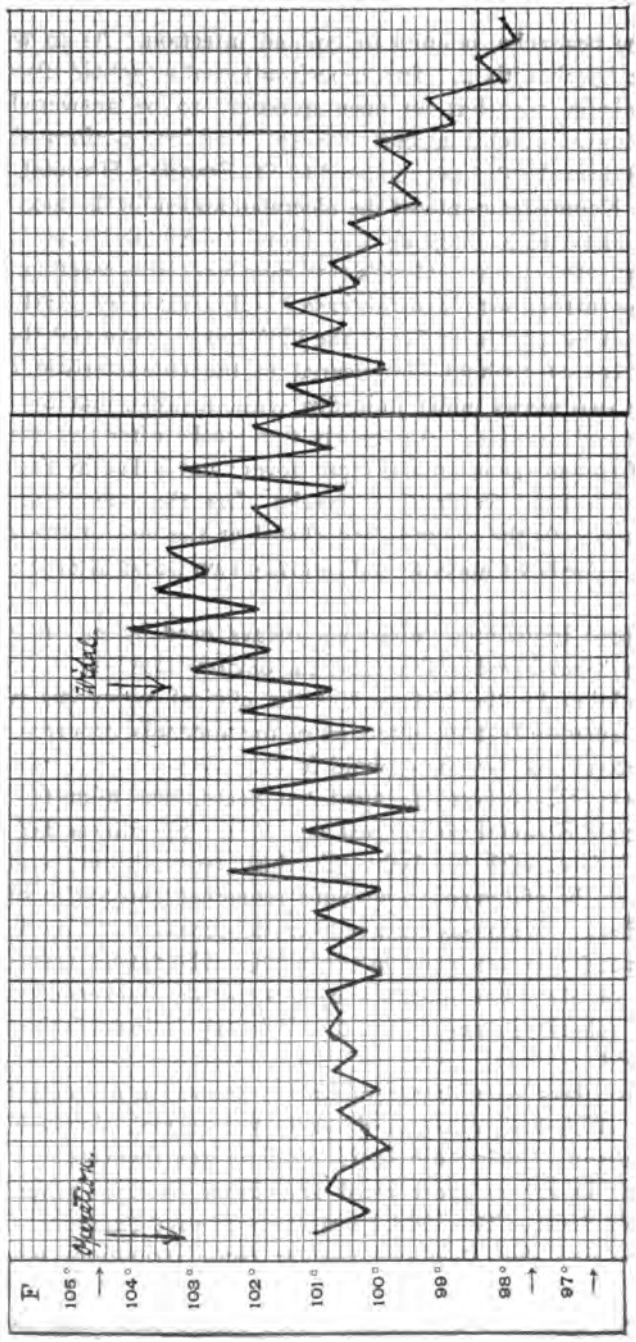

CHART OF CASE II.

assisted the patient to soon overcome the threatening general invasion of the typhoid bacilli.

In this second case, I believe that the appendix was the first locality in the intestine where the typhoid germs entered the mucosa. The rest in bed and the fluid diet after the operation somewhat retarded the more rapid extension of the typhoid process.

DISCUSSION.

DR. CAILLÉ.—I have never recognized this combination in the child. We know of its occurrence in adults and I have been much interested in the observation made by Dr. Seibert, and particularly in the diagnostic features of his cases. Appendicitis means an infected appendix and the infection is just as liable to take place from a typhoid gut as from any other.

DR. SEIBERT.—The diagnosis of typhoidal appendicitis is based upon a very high temperature, low pulse and the characteristics of appendicitis, verified by operation, and, with positive Widal reaction. The only question of interest to me was to know how many members of the Society have ever recognized typhoidal appendicitis in a child. We all recognize it in adult life. I said in my paper that these were my first observations, but I think we occasionally overlook this complication, because no doubt the inflammation subsides in most cases before perforation takes place.

CHRONIC PARENCHYMATOUS NEPHRITIS IN A CHILD TREATED BY RENAL DECAPSULATION. (EDEBOHLS' OPERATION).

BY AUGUSTUS CAILLÉ, M.D.,

New York.

Acute renal infection is occasionally so intense as to lead to a rapid and fatal termination. When renal tension can be relieved in no other way, an incision through the capsule of an acutely inflamed kidney is indicated and may establish a cure. Reginald Harrison, of England, has reported since 1896 a number of such cases and cures. The proposition to treat chronic nephritis surgically was first made by Dr. G. M. Edebohls, of New York, in April, 1899, after various observers, including Edebohls, Rose, Ferguson and Newman had noticed the disappearance of albumin and casts from the urine after operations upon the kidneys.

In the *Medical Record* on December 21, 1901, Edebohls reports 18 cases of chronic nephritis in adults treated by renal decapsulation. With a view of testing the efficacy of this operation in a severe and otherwise incurable case of chronic nephritis in a five-year-old child, I invited Dr. Edebohls to demonstrate his method and operate upon a patient who had been under observation for three years. The history of this case, briefly told, is as follows:

Clara Buchholz, five years old, had measles of a mild type at the age of two. One month later, her face, legs and abdomen were noticed to be swollen. A well-known physician diagnosed the case as one of acute nephritis from which she apparently recovered after four weeks. A similar attack a month later also ended in apparent recovery. On the occasion of a third attack, which was more severe, with suppression of urine of forty-five hours' duration, the child was admitted to the Babies' Wards of the New York Post Graduate Hospital, November 13, 1900. Her case was diagnosed as subacute nephritis and she was discharged improved December 30, 1900. On April 3, 1901, she was readmitted during my service with all clinical evidence of chronic nephritis: urine scanty and dark, containing albumin, all forms of casts and renal elements, blood and pus. Her eyelids were puffy and her abdomen contained fluid. After a course of sweat-

ing, dieting and the application of intestinal irrigation and the administration of diuretics and diaphoretics, she went home improved.

Nine months later, on February 1, 1902, I found her again an inmate of the Babies' Wards. She was generally edematous, her heart was markedly enlarged, the apex beat being at the sixth interspace, about an inch to the left of the nipple line. The heart sounds were clear, the second sound was accentuated. On the 15th of February, renal decapsulation was performed on both kidneys, the accompanying chart shows for one week before operation and for two weeks after operation a carefully kept daily record of temperature, pulse, respiration, stools, quantity of urine for twenty-four hours, its reaction, specific gravity, percentage of urea, and albumin and the character of casts and renal elements.

At the operation, both kidneys were found to be in a state of chronic parenchymatous nephritis. Each kidney measured over four inches in length and was three times larger in bulk than normal. They were delivered through the usual lumbar incision without much difficulty, the child being placed in a prone position with the abdomen resting upon an inflated round cushion. The capsules were split and removed and the kidneys were then replaced. The healing process was uneventful.

Regarding the rationale of the process, Edebohls in his article says: "Renal decapsulation, is performed with the object in view of creating new and liberal supplies of arterial blood to the diseased kidney. How does the operation effect this object and can the same object be effected in a better way? In answer to the question it is simply necessary to point out that both the denuded kidney and its fatty capsule are most liberally supplied with blood vessels; that both are brought together by the operation over the whole extent of the surface of the kidney and that the necessary result must be the formation on the most extensive scale possible of new vascular connections between the kidney and the fatty capsule embracing it. The fibrous capsule proper forms an almost impenetrable barrier to the passage of blood vessels between the kidney and its fatty capsule, as we have abundant opportunity to verify at operations upon the kidneys. It is not at all uncommon, for instance, in operating upon a kidney to find the blood vessels of the fatty capsule greatly increased both in number and size,

generally as the result of a perinephritis. Now and then a large artery of the fatty capsule will be seen entering and apparently penetrating the capsule proper. On raising the capsule proper from the kidney, however, the artery is not severed and further investigation shows that it does not enter the kidney but is lost upon and in the capsule proper, which has thus intercepted a possible new blood supply to the kidneys."

REMARKS.—During my professional career, extending now over a quarter of a century, I have made the following observations regarding kidney infection: Nephritis following acute infectious disease in children has a tendency to complete recovery. Children who survive the acute stage but continue to show albumin and renal elements for several months, also frequently make a complete recovery. In a certain percentage of cases, recovery does not take place. Seven of my patients who developed nephritis following infectious disease, such as scarlatina, diphtheria, measles, etc., under my personal observation, subsequently died in consequence of kidney insufficiency. One at the age of five, one at six, two at ten, one at fifteen, and two at the time of the first confinement after marriage at twenty-one and twenty-two. In view of the uselessness of medication, in chronic nephritis, the proposition to treat chronic Bright's disease, surgically, should be met without prejudice. The case which I report upon is not cured at the time of the present writing. The condition of the child when last examined is as follows:

Temperature, 99. Pulse, 90. Respiration, 20. The child is somewhat anemic but has gained in body weight. There is no edema. The urine shows a small amount of albumin and a few leukocytes and casts. The percentage of urea is 3.3 which is rather high. (See chart.)

From my observations in this case and in other cases which have come under my observation, I should be willing to advise inspection of the kidneys through lumbar incision in cases in which an acute nephritis, not secondary to heart lesions does not clear up in a reasonable time, say six months, and would furthermore advise decapsulation of one or both kidneys should they appear swollen and enlarged, with the hope of preventing acute nephritis from becoming chronic. I propose to keep this child under observation and hope to be able to report an improvement of its condition at our meeting next year.

MONTH OF FEBRUARY, 1902.

Day of month..	6	7	8	9	10	11	12	13	14	15	Operation.	16	17	18	19	20	21	22	23	24	25	26	27	28	Three Months after Operation. May 20
Temp. A. M. 6....	99[6]	98	98[2]	98	98	97[6]	98	98	98[2]	98[2]		98[4]	98[2]	98[2]	98	100[4]	100	98[2]	97[8]	97[6]	97[8]	97[8]	97[6]	97[4]	~ 99
Temp. P. M. 6....	99[6]	99[6]	99[4]	99[6]	98[6]	99	98[6]	100[2]	99	99		100[4]	100[4]	98[4]	100[2]	100[6]	101	99[6]	99[8]	99	99	99	99[6]	99	
Pulse A. M. 6....	100	104	96	90	90	90	92	90	92	104		92	96	98	90	116	120	96	100	96	90	92	92	90	~ 90
Pulse P. M. 6....	120	96	96	96	96	96	96	98	96	96		102	02	90	122	120	96	96	102	96	104	96	96	96	
Resp. A. M. 6....	28	24	24	24	24	24	28	24	28	28		24	24	24	24	28	28	24	28	28	24	24	22	24	~ 20
Resp. P. M. 6....	24	24	24	24	24	24	28	28	26	26		24	24	24	34	30	30	24	24	24	24	24	24	24	
Stools number, 24 hrs.	iii	ii	ii	ii	ii	ii	iii	ii	ii	i		ii	i	i	i iii	v	v	ii	i		ii	i	i	i	
Urine Quantity, 24 hrs.	ʒ xxv +	ʒ xxviii	ʒ xxxiii	ʒ xxiii	ʒ xvii	ʒ xxvi	ʒ xxii	ʒ xviss	ʒ xxii	ʒ xviii		ʒ xxvi	ʒ xiv	ʒ xxiii	ʒ xiv	ʒ vi	ʒ xi	ʒ xiv	ʒ ix +	ʒ xxiv	ʒ xxv	ʒ xvi	ʒ xx	ʒ xiv	ʒ xxii
Reaction	ac																								ac
Spec. grav.	1018	1020	1019	1018	1010	1014	1015	1022	1017	1020		1018	1018	1035	1033	1026	1029	1020	1016	1015	1012	1014	1016	1021	1027
Urea	1.4	1.1	1 3	1.9	1.2	1.4	1.7	1.9	1.5	1.4		1.5	1.8	1.8	2.8	2.1	2.8	2.1	1.5	1.15	1.00	1.1	1.5	2.6	3.3%
HNO_3	20%	25%	20%	20%	10%	10%	15%	10%	15%	15%		10%	10%	15%	20%	15%	15%	15%	15%	15%	15%	15%	15%	20%	
Albumin quant.	.1	.1	.15	.1	.1	.075	.1	.1	.1	.1		.075	.075	.1	.2	.2	.15	.15	.075	.075	.1	.075	.1	.075	.075%
Renal casts	Occasional hyalogr. Numerous bacteria.	Occasional hyalogr. Amorph. urates.	Same.	Same.	Same.	Same	Same.	Occasional hyalogr. Trace epithel.							Many hyaline. Many pus.	Same and waxy.	Same.	Gran. hyaline.	Same.	Same	Same.				Chlorid 1.7%. Leukocytes few. Renal epith. "

Occasional renal cells.
Few pus cells. Rarely a red blood cell.

Hyaline and granular casts abundant.
Waxy, epitheliated and pus casts occasional
Occasional red blood cells and pus cells.

CHART OF CASE OF RENAL DECAPSULATION.

DIPHTHERIA, WITH AND WITHOUT ANTITOXIN; 159 CASES.

BY CHARLES GILMORE KERLEY, M.D.,

Lecturer on Diseases of Children, Polyclinic Medical School and Hospital; Attending Physician, Infant Asylum; Assistant Attending Physician, Babies' Hospital; Physician Out Patient Department, Babies' Hospital, New York.

This subject is not brought before you with the idea of proving the value of antitoxin in diphtheria but rather to establish certain principles of action which should govern us in the use of antitoxin so that it may be of value to the profession at large.

One hundred and fifty-nine cases of diphtheria comprise the number of cases of this disease seen by me. Forty-two were operative (intubation) cases. One hundred and three were treated without the use of antitoxin and 55 with antitoxin. Among the first series 60 died and 43 recovered. Six succumbed in less than 48 hours and perhaps would not have been saved by our present methods of treatment. The duration of the disease in the remainder was from 48 hours to three weeks. The 103 cases occurred in New York Infant Asylum—an institution advantageously situated in the open country, built on the dormitory plan, and among children who were in better physical condition, better fed, housed and clothed than many of the patients who make up the antitoxin cases to be referred to later. Further, when ill, resident physicians were in attendance, seeing the patients many times a day, under the advice of the attending physicians; the patients were cared for by trained nurses, in buildings set apart for the exclusive use of the sick, and supported by a generous board of managers who supplied every need. The fact that these were institution children does not, to my mind, bear on the mortality except that it would tend to make the death rate lower.

The ages of the patients ranged from six months to five years. It may be interesting in this connection to call attention to the immunity of the great majority of the very young to diphtheria. Our youngest patient was four months old. The period covered by the epidemic was three years. There were constantly in the institution from seventy-five to one hundred infants from three weeks to six months of age, equally exposed with the older inmates, and but one took the disease.

Forty-eight of the fatal cases developed laryngeal symptoms and in thirty the stenosis was sufficiently severe to require intubation. Twenty-two cases had complicating nephritis. At the autopsy 53 cases showed membrane in the larynx, trachea or bronchi. In 5 cases the membrane extended to the fifth division. In 5 cases no symptoms had been present during life to suggest the presence of a small amount of membrane in the trachea. In one only the membrane was found in the esophagus, completely lining and adhering to the tube. Forty-six cases showed bronchopneumonia at the autopsy.

RECOVERY CASES, WITHOUT ANTITOXIN.—The youngest recovery case among the forty-three was four months of age; the oldest, ten years. Nine had laryngeal stenosis; four were intubated. The tube being worn respectively five, seven, thirteen and seventeen days. Diphtheritic paralysis occurred in five. These cases were treated by the methods then in vogue, internally with calomel, bichlorid of mercury, iron, chlorate of potash. Locally, with medicated steam inhalations, vaporized calomel, applications to the parts of trypsin, sulphur, solutions of bichlorid of mercury, chlorin water, etc. The only service we rendered these patients was by supportive measures. Suitable clothing, well ventilated rooms, easily digested nourishment, and judicious stimulation were provided, and then the child died or recovered depending upon the severity of the disease and the resistance of the patient.

After living with the above one hundred and three children, ill with diphtheria, seeing them several times a day, and in many instances administering the treatment myself, I am convinced that not one case was influenced favorably in the slightest degree by any *specific* treatment.

THE ANTITOXIN CASES.—Fifty-six cases comprise the antitoxin group; the ages of the patients ranged from nine months to ten years. Forty-one nonoperative cases with one death;

15 operative cases with three deaths. All showed the Klebs-Löffler bacillus.

Nonoperative, first day injection cases, fourteen in number, 12 cases received 2,000 units. In one only the dose was repeated the second day of illness. The throats of these were clear of membrane in thirty-six to seventy-two hours after the first injection. Two received 3,000 units. The throat in one was clear in thirty-two hours, the other in 120 hours after the first injection.

Nonoperative, second day injection cases, nineteen in number; 14 cases received 2,000 units, which amount was repeated in four on the third day. Five cases were given 3,000 units, and in one the amount was repeated on the third day. In 18 cases the throats were clear in from thirty-six to ninety-six hours after the first injection. One required five days before the throat was free of membrane.

Nonoperative, third day injection cases, four in number. One was given three injections of 2,000 units at twenty-four hour intervals. One, two injections 2,000 units at twenty-four hour intervals. One, two injections 3,000 units at twenty-four hour intervals. In two the throats were clear in seven days. In one case the history fails to mention when the membrane disappeared.

Nonoperative, fourth day injection cases. One case, 2,000 units were given, the throat cleared in forty-eight hours after injection.

Nonoperative, fifth day injection cases. One case, 2,000 units. Throat clear in 420 hours.

Nonoperative, eighth day injection cases. One case, 2,000 units which amount was repeated in twenty-four hours. Throat clear in seventy-two hours after first injection.

Two of the above had received immunizing doses. A child of nine months was given 300 units and developed diphtheria in four days. The patient recovered after one injection of 3,000 units. A boy four years old was given 1,000 units for immunization; he developed diphtheria in thirty-six hours which was controlled by one injection of 3,000 units. The throat cleared in forty-eight hours after the second injection.

Four patients developed diphtheritic paralysis of a mild degree. In two the muscles of deglutition were involved; in two, one lower extremity.

None of the cases had nephritis; the tonsils were involved in 136 cases out of the entire number of 159.

ANTITOXIN RECOVERY CASES—OPERATIVE.—Twelve in number, aged one to six years. *Second day injection cases,* six in number. One received 1,000 units. One received 2,000 units. Two received 3,000 units, and the third day 3,000. One received 3,000 units, throat clear in three days. One received 2,000 units, throat clear in three days; relapse on eighth day, 1,500 units given; throat clear in three days. *Third day injection cases,* six in number. Two received 3,000. Two received 2,000 units. One received 2,000 units.

One received 3,000 units and 2,000 units on fourth day.

Five were intubated on the second day of illness. Seven were intubated on the third day of illness. The tubes were worn from three to seven days in 10 cases. In one the tube was worn two weeks and in one it was worn four months.

ANTITOXIN CASES—FATAL. OPERATIVE.—Three in number. Ages—two, two years; one, three years. One case was injected the second day with 2,000 units, intubated the second day of illness and died the same day with diphtheria and lobar pneumonia. An early case and not enough antitoxin given.

One case was given 3,000 units on the fifth day, intubated on the fifth day, died in twelve hours.

One case was intubated the fifth day, 3,000 units given the fifth day, which amount was repeated twice at twelve hour intervals, the child died of heart failure in forty-eight hours after the first injection. One case died with pneumonia, the other two were injected on the fifth day. Both were septic at the time and practically hopeless.

Nonoperative.—My first and only fatal nonoperative case treated with antitoxin was given 1,000 units on the fourth day of the illness; the child died on the eighth day. These fatal cases do not in any way bear upon the value of antitoxin as a remedy in diphtheria and it is hardly fair to include them in the antitoxin group. In the nonoperative case it was my first experience with antitoxin. I did not know how to use the serum and it was not of the high order of efficiency that we have at the present time.

The good results in the cases treated with antitoxin are to be attributed to the early use of the remedy. In suspicious cases I do not wait for the culture. By suspicious cases I mean visible

membrane. I inject and then take a culture. I have injected a few cases which did not develop diphtheria later. To the best of my knowledge the recovery cases are all living at the present time and never have shown any untoward results from the use of the antitoxin.

In addition to bringing the antitoxin into use at the earliest possible moment it is necessary to be able to know when more is required; this is a point not generally appreciated. According to my observation the beneficial effects are not evident until ten or twelve hours have elapsed. If after twelve hours no improvement at all is evident, I reinject. After improvement is noticed by a fall in the temperature and by the membrane taking on a ragged granular appearance, I wait twelve hours more to observe the further changes. If at this time the character of the membrane remains unchanged, or shows fresh deposit with perhaps a rise in temperature and increased prostration, the antitoxin is repeated.

The chart which I present demonstrates well the effect of antitoxin upon the disease as indicated by the temperature. Improvement was noticed after each injection but was not sufficient to control the disease until 9,000 units had been given, this being the amount of the antidote which the little girl required to combat the poison in her system. (See chart, p. 129.)

The death rate in diphtheria may be reduced to a very small percentage by the early use of full doses of antitoxin, not less than 3,000 units which should be given during the first twenty-four hours if possible. This amount is to be repeated without hesitancy if improvement is not promptly observed. When in doubt intubate; so when in doubt, inject. This I practice and teach. About 20 per cent. of the cases develop urticaria which is annoying for a few days. I have noticed no other unpleasant results from the use of the serum.

I would establish the following principles:—

With visible membrane inject at once, and take a culture.

In croup, inject if there is inspiratory and expiratory obstruction.

The patients should be seen at twelve hour intervals.

Reinject in twelve hours if the patient is not improved or if improvement is not marked.

If continued improvement does not follow reinject at twelve hour intervals until the membrane disappears.

Dosage, 2,000 units for a child under one year of age, the amount to be repeated if necessary.

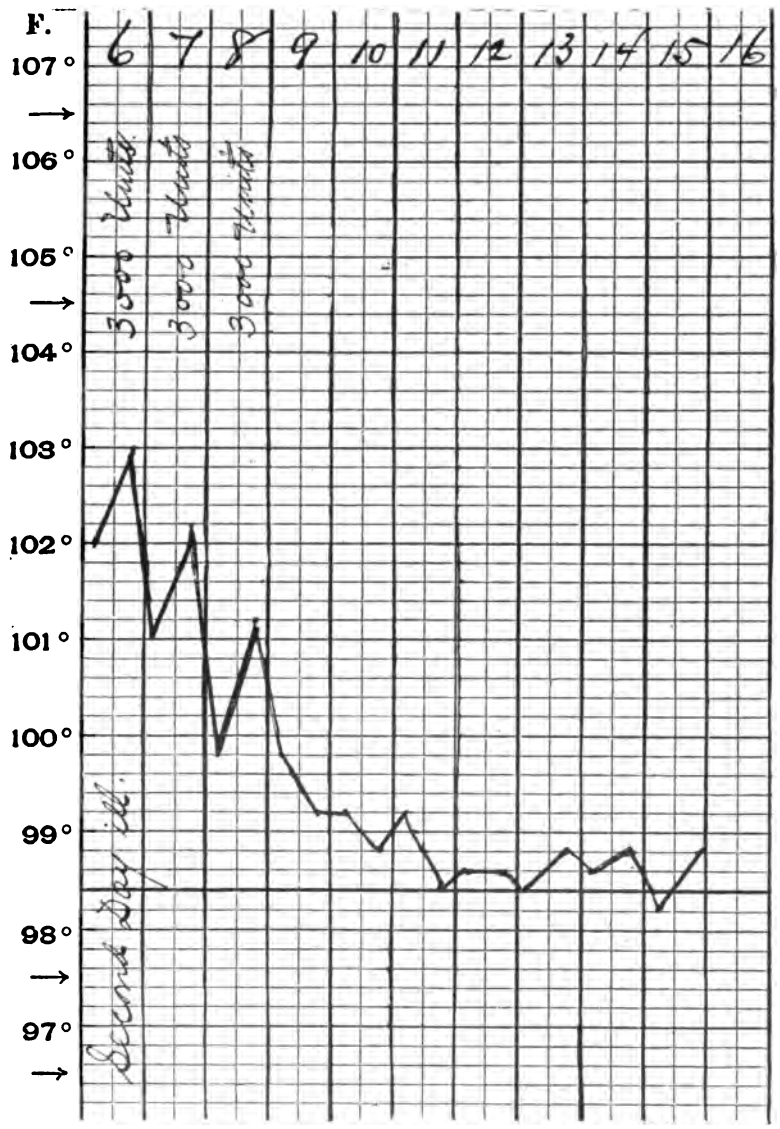

CHART SHOWING THE INFLUENCE OF ANTITOXIN IN REDUCING TEMPERATURE.

Three thousand units for a child over one year of age, the amount to be repeated if necessary.

SOME REMARKS ON INTUBATION IN DIPHTHERIA.

BY JOHN H. MCCOLLOM, M.D.,
Boston.

Before saying anything regarding intubation I should like to refer to the paper that has just been read. The paper was very interesting and I am glad that the point of early administration of antitoxin was mentioned. I do not know of anything that has been so forcibly brought to my mind as the importance of its early administration in doubtful cases. The physician has no right to wait for the result of a culture. Take a culture by all means, but if you wait twenty-four hours to administer the antitoxin you put the patient's life in jeopardy. In the past seven years at the South Department of the Boston City Hospital there have been 134 cases of diphtheria contracted by doctors and nurses, under the worst conditions, because the individuals were debilitated by hospital life. At the Boston City Hospital two doctors and four nurses lost their lives from diphtheria, before the days of antitoxin. In the 134 cases there was no death, because as soon as there were any symptoms of diphtheria the patients were given 4,000 units of antitoxin. These were cases of diphtheria both from a clinical and bacteriological standpoint. There have been instances where the young men and women have taken antitoxin unnecessarily, but there have never been any injurious effects following its use.

We are all aware that intubation is not a new thing. In 1858 it was brought before the French Academy of Medicine, but met with a poor reception and finally fell into disrepute. In 1885 Dr. O'Dwyer commenced his investigations and devised the tubes known by his name. We can all go back to the days before the use of antitoxin and all realize how hopeless we felt when called to a case of laryngeal diphtheria. Tracheotomy might be performed and the patient's life saved thereby, for a short time, but he would almost surely die from bronchopneumonia, or extension of the diphtheritic membrane, which extension was then not very well understood. One very important symptom indicating this extension is the formation of a tough, hard, tenacious mucus on the tracheotomy and intubation tubes. The death rate in tracheotomy cases was very carefully analyzed some few years ago by Drs.

Lovett and Morse, who tabulated some three hundred and twenty-five operations performed at the Boston City Hospital, and they found the death rate to be something like 75 per cent. Of course many of the patients were admitted to the hospital in a moribund condition. Intubation at that time was not done and it was not until 1889 or 1890 that the first case of intubation was performed at the Boston City Hospital.

Lately, Bayeux has published an extensive work on diphtheria and speaks at length on intubation. He has devised a set of tubes that he considers a vast improvement over the O'Dwyer tubes. It is a question with me whether they are such an improvement; but they do have one advantage, they can be easily extracted by what he calls his method of enucleation which cannot be done as well with the O'Dwyer tube as with the Bayeux tube. This is of some importance in private practice where you cannot always remain with the patient to extract the tube. I show you the Bayeux tube and you will see that it is short as compared with the O'Dwyer type. That is why it can be extracted with so much more ease.

One of its disadvantages is that it is more easily coughed up. You will notice that the obturator is attached to the introducer in a different manner from that of O'Dwyer's. In Ermold's modification of the O'Dwyer's set the obturator and introducer are all one piece. It is an interesting fact that in the Children's Hospital in Paris, in the pavilion named after Trousseau, the greatest opponent of intubation, all operative cases of laryngeal diphtheria are treated by this method.

The feeding in these cases is of great importance. Formerly I used the nasal method, but found that I was having too many cases of middle ear disease and so discontinued it and adopted the esophageal method. I had a tube of soft rubber devised for this purpose; it cannot become clogged.

Heretofore it has been considered that intubation was only applicable in children and that after fifteen years of age tracheotomy was indicated. But, having had some 6 or 8 cases of intubation in adults, I am convinced that the stenosis can be relieved easier by intubation than by tracheotomy. One of my patients was a man sixty years of age, in whom intubation was somewhat difficult, but less so than tracheotomy would have been.

Since the South Department was opened there have been 1,200

intubations and the death rate, including all cases, was 40 per cent. Eliminating deaths caused by diphtheritic paralysis, the rate was about 30 per cent. Eliminating moribund cases, patients dying within twelve hours after admission, the death rate was 25 per cent. I have looked over the statistics of the London and Glasgow hospitals for diphtheria and find the death rate to be 35 per cent. In these hospitals operations are not performed in apparently hopeless cases. Our death rate has been 25 per cent. and I think it is due to the fact that we operate early and give large doses of antitoxin.

Much has been said pro and con. about tracheotomy and intubation. In private practice, especially in the country, it is quite true that tracheotomy may be the safer operation, because the patient may cough up the tube, or it may become clogged, but in the hospitals or in the cities where the patient can have constant watching, intubation is much better for three reasons. First, very little shock; second, parents will consent to intubation much more readily, and third, the chances for bronchopneumonia are much greater in tracheotomy than in intubation, and the reason for this is readily understood; in intubation the air passes through the natural channel and the chances of irritation are comparatively slight. Baginsky in his work on diphtheria says that intubation does not predispose to bronchopneumonia.

It has been said that esophageal feeding is difficult, but our nurses at the hospital are able to do it with ease and I do not know of any accident that has ever occurred.

I know of only one instance where there has been a retained tube and that was the case of a little girl who coughed up the tube quite a number of times and then it became necessary to do a tracheotomy and she is wearing the tracheotomy tube now. I do not see how it is possible with care in performing the operation to produce any great injury to the air passages. The amount of force used should be slight.

So far as the treatment of laryngeal stenosis by other means, I have given up the inhalation of steam, sublimation of calomel and the croup kettle, because I find that they do not do good and I operate early.

The length of time a tube stays in is varied. I generally take it out at the end of three or four days. Sometimes I have to put it back, but not often. The most favorable thing that can hap-

pen is for the child to cough up the tube at the end of forty-eight hours and then have no return of laryngeal stenosis.

I want to say again, and want to emphasize the fact, that it is important to operate at once without waiting for the result of cultures, because otherwise very valuable time may be lost; and more than that the culture may be negative, for it is common to get negative cultures in laryngeal diphtheria. At least 50 per cent. of our laryngeal cases gave negative cultures. We had a patient a short time ago who had simply stenosis, the peculiar, difficult breathing that you see in laryngeal diphtheria. Cultures taken were negative. I intubated the patient and the tube stayed down two minutes when it was coughed up and with it a cast of the larynx and right and left bronchi. We had lately a patient who did not have any sore throat. The child had an attack of coughing and the attendant found on the bed a cast of the larynx. I immediately admitted the child to the South Department and he coughed up another cast at the end of twenty-four hours. After autopsy the lungs were put in formalin and on section you could see the minute bronchi filled with the diphtheritic membrane.

DISCUSSION.

Dr. Caillé.—I was very much pleased when I saw the announcement of this subject. It seems to me that in the last few years, since we know something about diphtheria, and since we are able to cure it, we do not discuss it as much as formerly. As a rule medical practitioners are prone to discuss things they do not know much about and cannot cure.

I agree fully with what Dr. Kerley said about the treatment of diphtheria—by antitoxin—and that our former treatment was of little moment. Still I would remind him of one fact, that before we had antitoxin, our results were better with croup cases when we used mercury before intubation than when it was not given. It seemed to have some effect, probably in loosening up the membrane. Furthermore local disinfection of the nasopharynx will always be of considerable benefit. My personal experience is fully in accord with what the reader of the paper has said about the management of his cases and I have nothing to add to his excellent presentation of the subject.

I also want to endorse every word that Dr. McCollom has said on the subject of intubation. In croup my management is *antitoxin* early and intubation when necessary. I have used all the instruments on the market and still prefer the old O'Dwyer tubes. The short tubes are coughed up and in private practice this is

very annoying, for sometimes we are not within call. O'Dwyer recommended short tubes in cases of foreign body in the trachea when tracheotomy could not be done.

Intubation in adults has been less satisfactory in my hands. I was once asked to tube an adult in the office of a colleague, and did not recognize that there was subglottic obstruction. The patient became cyanotic and after I had introduced the tube stopped breathing when I had removed the tube. I immediately did a tracheotomy and subsequently found that I had struck a subglottic, cauliflower growth and that the patient was bleeding into the lung. The hemorrhage was stopped and the patient recovered. Unless I am absolutely sure that there is no obstruction of this character I will not tube an adult.

A word about the retained tube cases. We have in the Babies' Wards of our hospital two or three retained tube cases every year. The obstruction is generally cicatricial and the result of injury in intubating. It would be well if this society were to express the opinion that intubation is not an operation that everyone can perform or should perform and that no one should attempt it without practice and experience.

Dr. Jennings.—I wish only to endorse the paper of Dr. Kerley. My observation covers a period extending from preintubation days, an experience of 400 operative cases. The last publication of my assistant, who does most of the work now, covers 230 operations, and since that time we have operated perhaps fifty times more, which would cover in the last three or four years nearly 300 operations, an experience sufficiently wide to enable one to form some conclusions. The mortality rate has been 23 or 24 per cent., the cases being almost uniformly in private practice.

With the dose of antitoxin it has been very rare that we have exceeded 6.000 units. From an economical standpoint the determination of the dose is of importance. While there seems to be no objection to enormous doses, still I think these large doses are not to be advised. Two thousand units repeated in six to twelve hours, perhaps three injections, puts an end to the diphtheritic process and our reasonable mortality rate would seem to show that this is a safe dosage.

In regard to the esophageal feeding, I must confess that while it is occasionally necessary, I rarely resort to it. Either the Castlebury method, or the plan of putting the head of the baby over the table and allowing it to suck the milk is better than the esophageal method.

I agree with Dr. Caillé, that the long intubation tube is to be preferred in private practice, as it is very annoying to have the tube expelled, and the shorter the tube the more liability there is of expulsion, so I cling to the old O'Dwyer tube.

Dr. Chapin.—We all prefer intubation but my experience goes

back to the time when I was beginning practice and we had a great many tracheotomy cases in the tenement houses. There was one thing constantly happening and that was the collection of thick, mucopus in the lumen at the bend of the canula. The nurse would take it out, clear the open ends and think that the canula was free and put it back, and, several times I came in just in time to save the child's life. With reference to pneumonia I found that by allowing the child to breathe through a moist sponge it could often be prevented; a sponge as large as my hand was first put in hot water and the nurse every fifteen minutes put this right over the tube so that the child would breathe warm, moist air. In that way a great many cases would get along after tracheotomy without developing pneumonia.

With reference to intubations, we have a great many at the Willard Parker Hospital and I must say I think the long tube much preferable. Even then the tube is often coughed up and the child nearly dies before the house physician can be brought into the ward. I would say, too, that we send out with our ambulance a man to intubate, which I think a great advantage. In 1 or 2 cases instead of using artificial respiration I have held the child up by the feet with the head hanging down. One case in particular I remember, where we had much difficulty in getting the tube in and where finally it was gotten in, but the child was blue and did not breathe, it seemed to us, for a great while; we simply held up the child with the head down and in about thirty seconds it recovered.

Dr. Buckingham.—I believe that intubated cases can often be fed by raising the foot of the bed and using soft solids and, I think the use of the esophageal tube is unnecessary cruelty where it can be avoided.

Dr. Cotton.—I would like to ask Dr. Kerley and Dr. McCollom if they have not found, since the use of antitoxin, in its well-known effect of loosening up the edges of the membrane, that the tube will sometimes catch the loosened membrane and push it down and cause occlusion of the lumen of the tube. In an experience of four years as city physician in Chicago, diphtheria being quite prevalent at the time, and two years in charge of the infectious wards of the Cook County Hospital, I had some opportunity to watch these cases. I found that in intubating the larynx I met with this difficulty more frequently after than before we used antitoxin, and this I attributed to the use of the antitoxin in loosening the membrane. I issued an order to the house physicians that they should never proceed to intubate without having laid out the tracheotomy case and in several instances I am satisfied that this precaution saved the life of the child.

Dr. Crandall.—The point referred to by Dr. Cotton has struck me too. Where there is very loose, soft membrane it fills

up the tube and separates in pieces that obstruct the tube in a very troublesome manner. In one case under my care the tube had to be taken out repeatedly.

Dr. Chapin.—Does Dr. McCollom regard a retained tube as always evidence of traumatism in introduction?

Dr. McCollom.—Not always, of course. It may be due to postdiphtheritic paralysis, paralysis of the cords. That occurs perhaps in about 1 per cent. of cases. Where the child retains the tube for weeks only that is not what I mean by retained tube, but where it is retained for months. I think a long retention is due to trauma. You can see by means of the mirror when there has been injury to the cords.

Dr. Adams.—In a case of intubation do you feed by the esophageal method?

Dr. McCollom.—Yes, in the majority of cases.

Dr. Kerley.—I have fed with the tube but only when sufficient nourishment could not be given by other means; with the head lower than the trunk and turned a little to one side I usually succeeded with spoon feeding. Tube feeding has one advantage, however: We know just how much nourishment the child is taking. There is one other measure which should be mentioned because of the relief it affords the patient. I refer to normal salt solution irrigations of the throat; 110° is the usual temperature. Irrigation is best carried out with the fountain syringe, the child holding the tube between the teeth, allowing a fall of about two feet for the solution to flow into the child's mouth and out again. The relief afforded is very great as I can personally testify. I never employ nasal irrigation on account of the danger of forcing infection into the eustachian tubes.

Concerning the string in intubation I always leave it attached to the tube until the child is through coughing, which is generally about fifteen to twenty minutes.

Regarding Dr. Cotton's question about pushing detached membrane down before the tube I had only 1 case where a child stopped breathing and on removing the tube I found it plugged with membrane.

I do not believe that the cause of the so-called retained tubes is always a traumatism. In my case there had been no difficulty whatever in putting in the tube. The dyspnea with the tube removed was entirely inspiratory. There appeared to be an abductor paralysis. I think in not a few cases injury to the parts is caused by the tube not fitting.

I wish to emphasize the importance of repeating the dose of antitoxin, that one injection must not be relied on, that there can be no rule as to the amount which a patient will need, upon the degree of the poison depends the amount of the antidote required.

THE MANAGEMENT OF THE FAT PERCENTAGE IN FEEDING DIFFICULT CASES.

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The present-day effort to simplify methods of infant feeding, while commendable from the point of possibly interesting many practitioners who have hitherto been unable to understand the percentage system, nevertheless fails to bring into play the wonderful flexibility of this valuable instrument of precision—the very feature that renders it so infinitely superior to older methods of combining milk fluids.

In feeding ordinarily healthy infants, almost any combination of the milk solids within the average limits of dilution may prove successful, and thus ratios of fat to proteids corresponding to 4 to 1, 3 to 1,.2 to 1, or 1 to 1, may give excellent results in a large number of normal cases.

Unfortunately, the pediatrist is much more frequently called upon to feed abnormal infants, or those whose digestive organs have been seriously damaged by previous disease or unskilful efforts at early feeding, and it is here that the more delicate manipulation of the percentages of the solids becomes an absolutely indispensable aid in diet regulation, and not the mathematical fad of a visionary theorist.

Since relative underfeeding must be the keynote of the dietetic treatment of all these cases, it stands to reason that in feeding any given infant the largest amount of nourishment possible under the conditions will be secured when the percentages of fat, proteids, and sugar of the food mixture closely approach, but do not exceed, the highest measure of digestive capacity for each of these elements. If 1 per cent. of proteids is easily digested by a certain infant, it may be possible that 1.10 or 1.15 per cent. also is readily assimilated, while 1.20 per cent. may prove too high, as shown by the production of indigestion when the proportion of proteids is raised to this figure. It is there-

fore evident that a proteid percentage not much above 1.15 expresses for the time being the highest measure of the dilution of proteids which this infant can digest. In the same way the limit of perfect fat digestion may be found at about 1.75 per cent. It is therefore reasonable to conclude that a formula containing 1.75 per cent. of fat and 1.15 per cent. of proteids represents for the time the highest proportions of these two elements of the food which this infant can digest at one meal. It is quite evident, therefore, that simple integral ratios of fat to proteids, like 4 to 1, 3 to 1, 2 to 1, or 1 to 1, offer very inadequate combinations when the natural ratio for any given infant varies so greatly from these simple proportions, and especially when every hundredth of 1 per cent. of either element that could be added represents just so much more food for the starving tissues.

Such infants, with very few exceptions, are the subjects of a more or less severe chronic catarrhal inflammation of the whole, or of some limited part, of the gastrointestinal mucous membrane. Of these, the most frequently occurring and the most difficult to manage are the cases of chronic gastric catarrh, or of chronic catarrhal irritation of some limited area of the upper portion of the small intestine, to which the term chronic high intestinal catarrh may be applied. In some of these latter cases the catarrhal lesion is so close to the stomach that it is often with difficulty distinguished from an essential gastric catarrh, and in some, indeed, the two lesions are in all likelihood associated by continuity of structure. It is impossible at the outset of treatment in any particular case, accurately to estimate the degree of damage that has been sustained by the affected mucous membrane. Some cases that at first sight appear to be dependent upon serious involvement of the mucosa, improve quickly and regain health after a very short course of treatment; others that seem to promise rapid improvement show by their subsequent progress that the structural damage has been great, and only after months of carefully adjusted underfeeding, during which disheartening periods of relapse or standstill will occur, can a cure be effected and a perfectly satisfactory diet reached.

In all such cases recurring pain after feeding, persistent vomiting or regurgitation of more or less considerable quantities of food soon after the nursing, and the frequent occurrence, dur-

ing the earlier stages of treatment, of acute exacerbations of catarrhal irritation—these, together with wasting, anemia, constipation, and marked debility, constitute the chief clinical features of this serious condition which is so familiar to us all.

According to the commonly accepted standards, regulation of the diet in such cases chiefly confines itself to efforts to reduce the proteid percentage to such a point as to prevent the appearance of curds in the stools, while attention is but rarely directed to a study of the more careful regulation of the fat percentage. Holt is the only writer upon infant dietetics of recent years who has clearly pointed out that too high a fat as well as too high proteids may be a source of trouble in these cases. There is a distinct danger, however, that in reducing the percentages of both fat and proteids we may reach a combination of milk solids, which, while admirable from a theoretical point of view, nevertheless offers a food mixture entirely too weak to supply a sufficient amount of nourishment to the already starving tissues of the infant. It is with the purpose of suggesting a remedy for this serious defect of low-feeding mixtures that I venture to describe a method by which this deficiency in the proteids may be largely remedied, while at the same time the necessary reduction in the fat and caseinogen percentages may be maintained as long as the requirements of the case demand.

While the simple rules for regulating the fat percentage are readily applied in ordinary cases of feeding, it often becomes a question of considerable difficulty to recognize the disturbing influence of too high a fat in cases in which vomiting is a constant feature of the disease—cases in which the stomach has become so irritable that the slightest interference directly or reflexly acts in provoking vomiting of food at any time during the intervals of feeding. In the severer cases of chronic gastric or chronic high intestinal catarrh a certain amount of regurgitation after many of the feedings must be expected for a considerable time, a fact which in conjunction with necessarily low percentages in the feeding mixture explains the slow progress of these cases toward recovery. If, however, a careful regulation of the proteid percentage be not followed by prompt subsidence of pain and by distinct lessening of the vomiting, the influence of too high a percentage of fat should at once be suspected.

While it is quite certain that many of these infants can satis-

factorily digest as much as 2, or even 3, per cent. of fat early in the course of treatment, it is equally true that in the more obstinate cases of chronic vomiting a temporary intolerance of any but the very lowest proportions of fat is a factor demanding adequate recognition, lest the mistake be made of ascribing the difficulty to the proteids, and in consequence subjecting the proteid percentage to still further, though unnecessary, reduction. Whether this intolerance of ordinary proportions of fat is to be attributed to idiosyncrasy, or to temporary deficiency or inactivity of the hepatic or pancreatic secretions, I have been unable to determine. I believe that either of these factors, or both, may be at times operative.

It is a curious fact that there seems to be in certain delicate infants a distinct inability to digest cream, when added even in very small proportions to a milk dilution. In several cases that have recently been under my care, it has been impossible in the earlier months to increase the fat percentage of a milk dilution by adding cream in any appreciable amount—in one case, five to ten drops of cream added to a bottle of diluted milk at once producing pain, vomiting and flatulence, with disturbance of intestinal digestion and curdy masses of fat in the stool; and the same experience has occurred with certain infants temporarily fed, for sufficiently good reasons, upon condensed milk, when an effort was made to increase the very low proportion of fat in such a mixture by adding small amounts of fresh cream, as, for instance, half a dram of cream to a bottle of three or four ounces. In fact it seems to me quite probable that the low proportion of fat in condensed milk solutions constitutes one of the factors, in conjunction, of course, with low proteids, that sometimes makes this preparation readily tolerated in certain cases in which more reasonable modifications fail—an experience that I doubt not has occasionally occurred to the mortification of others beside myself.

Moreover, it has been my experience to encounter children of more advanced age who seemed to digest satisfactorily a partly skimmed milk, but suffered from frequent digestive disturbances when whole milk was steadily taken. In one marked instance of this kind, the mother gave the history that in infancy this child could not stand the addition of cream in any propor-

tion to a milk dilution, which was readily tolerated without cream.

I have been at a loss satisfactorily to account for this apparent intolerance of cream, for in the infantile cases that came under my own observation in private practice the greatest care had been exercised to use a cream derived from the same milk by gravity raising, and therefore of the same degree of freshness. Whether the fact pointed out by Freeman, that cream contains a considerably larger number of bacteria than the under milk from which it separates is of any importance in this connection I am unable to decide, but certainly no evidences of gross infection could be noted in the stools, and several experiments made on different days were always followed by similar results. The most rational explanation would be that the additional fat contributed by the cream served to raise the total fat percentage above that which could be normally digested at the time. Some credible support for this hypothesis is given by the fact that later on in the course of these cases the fat percentage could be cautiously raised by beginning with very minute additions of cream and gradually increasing the amount as tolerance was established; and this is not at all unreasonable when it is understood that the addition of one teaspoonful of a 16 per cent. cream to a bottle of three ounces increases the fat percentage by 0.64 of 1 per cent.—quite a considerable advance.

We have not yet attained sufficient skill in the differential analysis of the fat bodies of milk to determine whether the relative proportions of these bodies in the fat of cow's milk differ appreciably from those of the human milk fat, so that speculation on relative degrees of digestibility of the two varieties of fat would be at present unprofitable. Fortunately the practical requirements of the problem are satisfied by increased dilution of the cow's milk fat in cases that manifest an inability to digest proportions approaching those found in human milk.

Apart from this idiosyncrasy of some infants in being unable to digest reasonable proportions of butter fat, temporary weakness of fat digestion dependent upon feeble activity of the digestive juices is an important feature of all cases of chronic catarrhal irritation of the upper portion of the *prima via.* If required to classify the cases in which this weakness of fat digestion seems to be present, I should assign the most important

place to the cases of high intestinal catarrh, giving a much less prominent one to cases of pure gastric catarrh, and, naturally, almost as important a one to the cases in which the two lesions are associated. With very few exceptions, and these probably exclusively in the rare instances in which any form of milk food seems poisonous, the control of the proteid percentage has been greatly simplified by means of differential modification, as described in a recent paper on the subject (*American Journal of the Medical Sciences,* October, 1901). According to the relative proportions of the various percentages of fat, lactalbumin and caseinogen desired, these mixtures may consist of the following combinations of the milk fluids and water: simple cream dilutions; cream, milk and water; cream, whey and water; milk and water; milk, whey and water; cream and whey; milk and whey; cream, milk and whey; and cream, milk, whey and water. Each of these combinations has been employed under appropriate conditions with perfectly satisfactory results. In the great majority of cases the whey proteids seem to be digestible in any possible proportion, whether at a maximum close to 1 per cent., when combined with a very small fraction of 1 per cent. of caseinogen (*e. g.,* 0.10 per cent.), or correspondingly lower when higher percentages of caseinogen are used. Under a diet consisting largely of whey, the stools are usually a light golden brown in color, of a consistency varying from that of thick French mustard, when the caseinogen is low, to that of ordinary well-digested milk stools, as the proportion of the caseinogen is gradually increased; and in many cases in which the muscular tone of the abdominal walls is fairly maintained, natural movements once or twice a day are the rule.

According to this plan of modification the fat is supplied either by cream, cream and whole milk, whole milk alone, or a fat-containing whey. Whether whey carries any fat to the mixture will depend upon its being made from a fat-free separated milk or from milk partially or entirely unseparated. The calculations given in the paper just referred to are based upon the use of a fat-free whey; they may be easily readjusted for the use of a whey containing fat. Koenig's analysis of whey gives a percentage of 0.32 fat. In a number of analyses I have found that whey prepared from an ordinary 4 per cent. fat milk, without manual expression from the curd, draining away simply by

gravity, contains about 1.3 per cent. fat. This very respectable percentage of fat, which seems to be well tolerated by infants, renders this whey a more desirable ingredient of the mixture than that containing no fat, as made from a fat-free separated milk.

By the use of this fat-whey it is possible to obtain a mixture containing a fair percentage of fat without recourse to the addition of cream to the whole milk used to supply the caseinogen percentage. This seems to be of practical advantage in the cases that show intolerance of cream or of the ordinary percentages of fat.

In beginning the feeding of such a case, after thoroughly clearing out from the gastrointestinal canal the remains of recent milk feedings, a few feedings of egg-water are ordered, and these are followed by increasing proportions of whey, until the bottle consists of half whey and half egg-water. The percentage formula of such a mixture would be about 0.65 fat and 0.50 lactalbumin, neglecting the percentage of egg albumen which is immaterial to the problem.

The most rapid method of supplying increasing proportions of fat and caseinogen has been found to be the following. A milk and water, or cream, milk and water mixture of convenient amount, say six and one-fourth ounces (fifty fluid drams), is prepared, to which an appropriate amount of milk sugar or cane sugar may be added. For the cases found to have intolerance of rapid advances of the fat, as shown by the increased regurgitation and pain after the advances in the percentage, plain milk alone should be used in the milk mixture. Beginning with one teaspoonful, increasing quantities of the milk mixture are to be taken in combination with enough whey to make up the total quantity for the feeding.

In formulating the milk mixture the relative proportions of milk or milk and cream to the diluting water must be determined by the rapidity with which it is desired to increase the percentages of fat and caseinogen. Usually a proportion of about two parts of milk or various combinations of milk and cream to about one part of water offers a very satisfactory combination. Thus we may take about four ounces of milk and two and one-fourth ounces of water to make up the fifty drams; or one-half ounce of cream and three and one-half ounces of milk, or various

other proportions of cream and milk. It is quite evident that the fat percentage of such a mixture will vary considerably according to the proportion of 16 per cent. cream used, as is shown in the following table:

Cream,	0	oz.	Milk,	4	oz.	Water,	2¼	oz.	— F. 2.56; P. 2.56.
"	½	"	"	3½	"	"	"	"	— F. 3.52; P. 2.53.
"	1	"	"	3	"	"	"	"	— F. 4.48; P. 2.50.
"	1½	"	"	2½	"	"	"	"	— F. 5.44; P. 2.46.
"	2	"	"	2	"	"	"	"	— F. 6.40; P. 2.43.

or with a slightly lower proportion of the milk and cream:

Cream,	0	oz.	Milk,	3½	oz.	Water,	2¾	oz.	— F. 2.24; P. 2.24.
"	½	"	"	3	"	"	"	"	— F. 3.20; P. 2.21.
"	1	"	"	2½	"	"	"	"	— F. 4.16; P. 2.18.
"	1½	"	"	2	"	"	"	"	— F. 5.12; P. 2.14.
"	2	"	"	1½	"	"	"	"	— F. 6.08; P. 2.11.

It will be observed that the percentage of fat increases very rapidly in these combinations, while that of the proteids declines very slowly. The rapidity of increase of the fat must be determined upon while bearing in mind that a fair proportion of fat (1.3 per cent.) is present in the whey. Having decided upon the particular combination of milk and cream that will give a rate of increase of the fat commensurate with our estimate of the toleration of the infant, we begin the prescription for the bottle with one teaspoonful of the cream mixture and enough whey (sometimes at first part whey and the balance diluent) to make the desired quantity of the bottle: Thus

℞—Cream mixture f 3 i.
Whey f 3 xv.—M.

The calculation of the percentage value of this formula is readily made. The cream mixture contributes $^{1}/_{16}$ of its percentages while the whey contributes $^{15}/_{16}$ of 1.3 per cent. fat and 1.00 per cent. lactalbumin. The relative proportions of caseinogen and lactalbumin may be determined by taking $^{4}/_{5}$ of the proteid percentage contributed by the cream mixture for the caseinogen percentage, and adding the remaining fifth to the lactalbumin percentage supplied by the whey. In the above combination suppose the cream mixture was composed of one-half ounce cream and three ounces milk. The one dram of this in the two ounces of whey mixture would contribute 0.20 per cent. fat and 0.14 per cent. mixed proteids, the latter of which may be divided into

0.03 per cent. lactalbumin and 0.11 per cent. caseinogen. The 15 drams of whey would contribute 1.22 per cent. fat and 0.93 per cent. lactalbumin and the total mixture of the bottle thus contains fat, 1.42 per cent.; proteids, 1.07 per cent., consisting of 0.96 per cent. lactalbumin and 0.11 per cent. caseinogen. Two drams of the cream mixture and fourteen drams of whey would give fat, 1.54 per cent., proteids, 1.15 per cent., consisting of lactalbumin 0.93 per cent. and caseinogen 0.22 per cent.

In these combinations it will be noted that for each additional dram of cream mixture, the total quantity being kept constant at two ounces, the fat percentage advances 0.12 per cent., while the total proteid percentage is increased by only 0.08, or the fat increases one and one-half times as fast as the proteids. In many cases this rate will not be found to be too rapid at the start, and until a fair percentage of fat (2.50 to 3) is reached. But in the cases showing great susceptibility to fat increase it will be found that a slower rate of increase of the fat will be attended with less or no disturbance of digestion. When the cream mixture contains only 4 per cent. cream (whole milk), as shown in the second table, the relative rate of increase of fat to proteids would be as 0.06 to 0.075.

If whey from fat-free milk were used, the fat percentages would begin at a much lower figure (0.14) but the relative rate of increase would be as 0.14 to 0.08 since no fat would be removed by reduction in the quantity of whey. In the cases most intolerant of fat increase I am thoroughly convinced, as Holt has already stated, that a plain milk dilution offers a means of more rapidly increasing the caseinogen percentage than any other combination.

This fact was very suggestively emphasized in one of my experiences during the past winter. The baby, a boy, weighing about eight pounds at birth, was first seen at the age of seven weeks, after the usual history of unskilful feeding on all sorts of foods and milk mixtures. The weight then was seven pounds, one ounce. He was suffering from a high intestinal catarrh, with possibly some involvement of the stomach as well. The above plan of feeding was adopted, and while gain in weight, as shown by the chart, was steady from the start, it was found impossible to get him to take comfortably in a bottle of 2½ ounces

more than 3 drams of a cream mixture of 50 drams composed of cream 1¾ ounces, milk 2 ounces, and water 2½ ounces, containing percentages of 5.76 fat and 2.28 proteids. At this point the percentages of the bottle were fat, 1.97; proteids, 1.19, consisting of lactalbumin 0.92 and caseinogen 0.27. For fully two months the amount of cream mixture that could be taken remained about 3 to 3½ drams. Finally on the ground that 2 per cent. of fat was the limit of the fat digestion, a simple whole milk dilution was made, containing 2.56 per cent. of fat and proteids, and it was found that the number of drams of the milk mixture could be very promptly increased to 6 in a 3 ounce mixture, in which the caseinogen percentage had been advanced to 0.51 per cent., about double what it had previously been, the lactalbumin slightly declined to 0.88 per cent., and the fat suffered reduction from 1.97 to 1.61 per cent. Notwithstanding the increase of caseinogen and the decrease of fat, he gained more in the following two weeks than in any previous fortnight, as shown by the gains of each two weeks from the start; 1½ oz.; 5oz.; 7oz.; 11½oz.; 5½oz.; 7½oz.; 8oz.; 8½oz.; 1½oz.; and finally, for the two weeks following the change to whole milk, 13 oz. The quantity at a feeding was rapidly increased to 4 oz., 4½ oz., 5 oz., and 5½ oz., the same ratio of milk mixture to whey, 1 to 3, being maintained. At this point the baby had improved so greatly that another effort was made to introduce cream into the milk mixture, and in a short time the quantity was raised to 1 ounce. The milk mixture, which had been doubled and then trebled to permit of increasing quantities, consisted at last report of

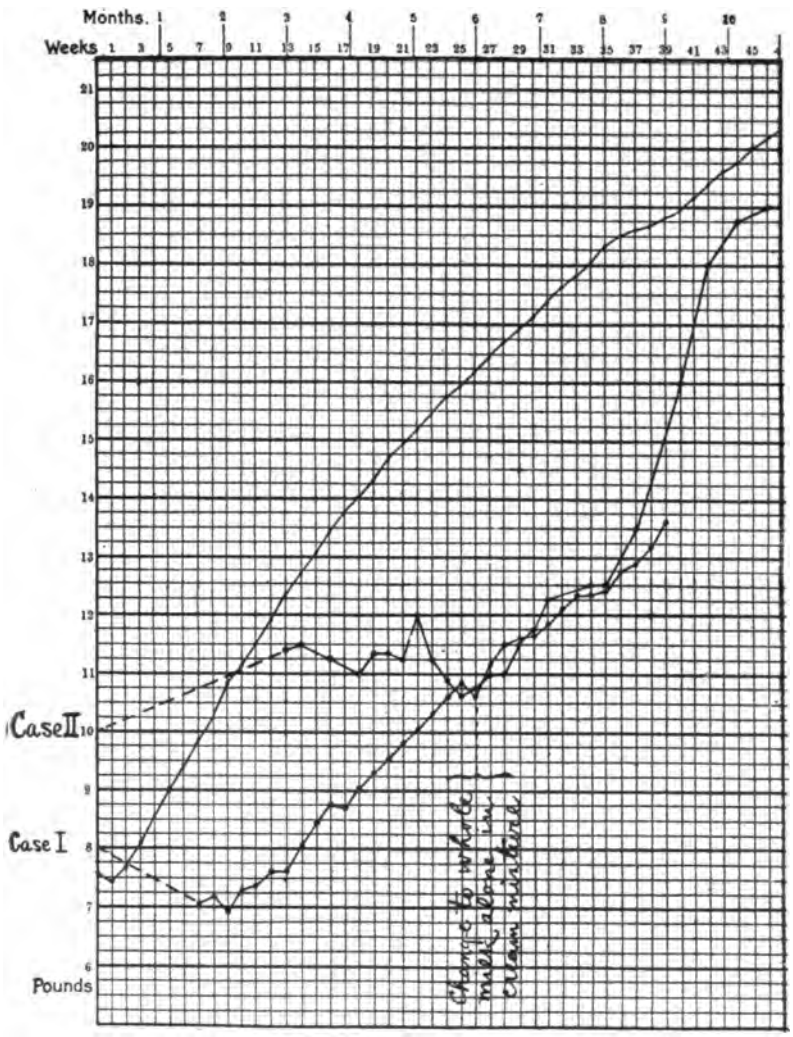
Cream,	1 oz
Milk,	12 "
Water,	5¼ "
	18¼ oz.

=150 drams, with percentages of 3.41 fat and 2.75 proteids. The bottle now consists of

Cream mixture,	2½ oz.
Whey,	3½ "
	6 oz.,

given every 3 hours; 7 bottles in the 24 hours, the resultant percentages of which are fat 2.18; proteids 1.73; lactalbumin 0.81; caseinogen 0.92. It is a curious feature of this case that quite recently another effort to increase the fat percentage above

2.18 has proved unsuccessful, showing how very close to 2 per cent. this baby's digestive factor for fat still remains.



WEIGHT CHART.

The sugar percentage has been taken care of by adding appropriate quantities of sugar from time to time, but reference to this has been omitted here, as having little bearing on the special points under discussion.

This has been one of the most trying cases of feeding I have ever encountered; the practical results, except as regards weight, which will now be rapidly made up, have been the most satisfactory. Despite the marked underfeeding which has been necessary until very lately, the average gain in weight has been steady and has averaged a pound a month, until recently when his teething has produced some disturbance and there has been a slight faltering in the curve. The proportions of food, however, are now being rather boldly increased and the baby seems ready to take it. At the present writing his weight at the age of nine months is thirteen pounds, one ounce; he is bright, active, of splendid color and firm flesh, though not yet very plump. He has already cut eight teeth, shows no signs of rachitis, has one movement a day, usually without solicitation, is constantly trying to stand on his feet and sleeps well all night, waking once for a bottle.

The weight-curve of a healthy infant (see chart, case II) fed according to the same method shows how rapidly weight may be gained upon a milk and whey mixture under more favorable conditions.

In conclusion I would emphasize two points that have been brought out:

1.—In cases of chronic gastric or chronic high intestinal catarrh, in which underfeeding for a considerable time becomes necessary, the most satisfactory results may be obtained by the use of whey mixtures, in which the deficiency of proteids in ordinary modifications is supplemented by a high percentage of lactalbumin.

2.—In many of these cases unusually low percentages of fat must be given, and for this purpose dilutions of milk without cream in conjunction with a fat-containing whey offers the most expeditious means of increasing caseinogen and of keeping the fat percentage within the bounds of digestive tolerance.

THE PROTEIDS OF MILK.

BY HENRY DWIGHT CHAPIN, M.D.,
New York.

At the meeting of this Society, held at Niagara Falls, in 1901, the writer incidentally mentioned an analysis of woman's milk in which the total proteids were stated to consist of casein 1 per cent. and albumin .5 per cent. At that time it was suggested that the proportion of casein should have been .5 per cent. and the lactalbumin 1 per cent. In other words, that the proteids of woman's milk consist of about one-third casein and two-thirds lactalbumin, Koenig's analysis being cited as authority.

Since then, in looking over many analyses of the milk of various animals, to see if there was any particular ratio between casein and lactalbumin, some interesting figures were obtained.

It appears that different methods of analysis have been used by different chemists, which accounts in part for the different ratios that have been published.

In some methods total proteids (N x 6.25) have first been determined, and then the total substance precipitated by cold dilute acetic acid, which is called casein. The difference between total proteids and the casein thus obtained has been called lactalbumin. In other methods the quantity of proteid precipitated by cold dilute acetic acid has been called casein and the quantity of proteid precipitated by boiling with acetic acid, after the casein was removed, has been called lactalbumin.

The wide differences in results and ratios between casein and albumin, reported by different chemists, have been the result of these differences in methods employed.

Van Slyke, who made a careful study of ratio between casein and albumin in cow's milk, made 106 analyses in triplicate of over 200,000 pounds of the mixed milk from 1,500 cows during a whole season, reported as late as 1893 that the albumin of cow's milk went as low as .56 per cent. and as high as .85 per cent., the aver-

age being 3.76 parts of casein for one of albumin. The albumin in all these analyses was determined by "difference." In 1894, with improved methods of analysis, the proteids of cow's milk, other than casein, were found to be albumin .28—.38, albumose .30—.50, only half as much albumin as previously reported.

In his report Van Slyke says: "I wish to call attention to the crude nomenclature in common use in stating the results of milk analysis for nitrogen compounds. It is an almost universal custom to call the total nitrogen compounds of milk casein. It would be quite as correct to call the fat of milk palmitin, or some similar name. The wrong use of the term casein leads to much confusion and it is highly desirable that we should use a more discriminating nomenclature. It is also desirable that in making analyses of milks, pains should be taken to separate and determine the different kinds of nitrogen compounds, since our knowledge of these compounds is far from complete."

In 1897, Babcock and Russell discovered a proteolytic enzyme in cow's milk, and subsequently in the milks of every species of animal that could be obtained. To determine the effect of this enzyme, the casein and albumin were separated by boiling with acetic acid, and the character and quantity of the remaining proteids were carefully determined. These proteids were classed as albumoses and peptones. From time to time the milks, that had been treated so as to prevent bacterial growth, were analyzed to determine the rapidity of the proteolytic changes, which were shown by an increase in quantity of albumoses and peptones. These changes took place slowly and the natural enzymes of milk, as far as can now be seen, have no practical significance. The analyses made in these determinations are of great value, however.

It was found that all milks contained greater or less quantities of albumoses and peptones, and quite contrary to the generally accepted belief, over *one-third* of the total proteid of the milk of six different women consisted of albumoses and peptones. The table herewith shows the exact quantity of total proteid and of albumoses and peptones in the different milks examined, and in a number of samples of cow's milk of different degrees of richness.

In the following analyses only the cow's and goat's milk were the mixed secretion of several animals.

	Total Proteid.	Albumoses and Peptones.	Other Proteids. By Difference.	PROTEIDS OF COW'S MILK. Total Proteid.	Casein.	Albumoses and Peptones.	Difference Albumin.
Sheep	4.75	0.58	4.17				
" Rapid Growers.	4.43	.62	3.81				
"	3.75	.62	3.13				
"	4.75	.56	4.19				
"	5.00	.62	4.38	2.87	2.12	0.50	0.25
Goat	4.87	.37	4.50	3.18	2.68	.25	.25
Pig	4.51	1.06	3.45	3.25	2.50	.31	.50
"	4.37	1.00	3.37	3.31	2.44	.31	.56
Mare	1.75	.56	1.19	3.43	2.87	.25	.31
Burro	1.54	.62	.92	3.43	2.87	.31	.25
Human	1.75	.62	1.13	3.50	2.81	.25	.44
" From 6 Different Women taken during 7 months.	1.75	.62	1.13	3.56	2.68	.31	.57
"	1.68	.62	1.06	3.62	2.94	.43	.25
"	1.68	.56	1.12	3.62	2.56	.50	.55
"	1.75	.62	1.13	3.6	2.75	.25	.68
"	1.68	.62	1.06	3.75	2.87	.25	.63
Half bred Buffalo	3.00	.25	2.75	4.50	3.50	.44	.56
Cow	3.18	.25	2.93				

It is possible that the cause of the difference in percentage of casein reported in woman's milk may be found in the following statement by Wynter Blyth, an English authority on food. "The easy separation of the casein and albumin by acetic acid and carbon dioxid only applies to the milk of cud chewers; with human milk, the milk of the horse or ass, the process gives no good results. When treated the same way the casein appears to coagulate, but it is in a state of such fine division that nearly all of it remains suspended in the liquid, and filtration through paper becomes impossible." It is thus quite possible that in the older analyses a portion of the casein of woman's milk has been classed as albumin.

It will be evident that until the *composition* of the different proteid bodies found in milks is discovered, and some uniform methods of separating them are agreed upon, comparing milks by analyses made by different methods may lead to erroneous conclusions. Whey and cream mixtures may not be such close imitations of mother's milk as have been supposed. At present the comparison of milk analyses for proteid is of little use except by showing total proteid and thus the potential food value. The primary object in infant feeding is to increase the number of cells in the body. These all contain phosphorized proteids. Albumin does not contain phosphorus, so, theoretically, it is not

fitted for the sole proteid of a diet for any animal. A dog will die of starvation if fed on albumin alone, or in combination with fats and carbohydrates. Albumoses and peptones are transformation products derived from any form of protein, and are not such distinct bodies as neucleo-albumins or albumins. With the present limited knowledge, it is a question whether it is wise to attempt to introduce into practical infant feeding methods that call for mixtures of different forms of protein supposed to exist in milk.

In some material, which will be published shortly, I expect to show that the differences between the milks of different species of animals are due to the fact that each milk is highly specialized, having functions beside simple nutrition, and that in the study of substitute feeding this fact must be recognized.

DISCUSSION.

DR. CHAPIN.—I think it is a common fault to call simply for creams. Now cream is one of the most inconstant things in the world and unless you have a laboratory with a centrifugal machine for the separation you are never certain about the cream. I have had commercial cream assayed and found that it may run 15 per cent. butter fat, again 18 and again 20. Two years ago I presented a method by which you can get a cream of known strength at home. It is based upon the "deep setting" process. The cream is put into a cylinder and allowed to stand for a number of hours, when nine-tenths of the butter fat will be on top. You must take all the cream and a certain portion of skim-milk, and then you get a cream of pretty constant strength. You do not care whether you are dealing with rich milk or poor milk, in the cylinder you get nine-tenths of the fat on top. The physician, then, instead of directing the mother to buy so much cream, tells her how to get it. The first ounce is taken out of the bottle with a teaspoon and then the ounce dipper, devised by me, is used. When the deep setting process has taken place you can tell it by the cream line. Each ounce has a certain fixed relation between the fats and proteids. The cream from deep setting is a great advantage over the commercial cream. I have given the instrument maker formulæ which he sells with the dipper. I find out how much the baby can digest and then figure the percentages. We must first find out how much the baby *can* digest, not what it *should* digest. I think we have been too theoretical because the babies vary so much in their digestive powers.

DR. WESTCOTT.—How do you make your calculation?

DR. CHAPIN.—If I want to have 3 to 1, for instance, I take the 12 per cent. cream; there I have fats 12, proteids 4 and sugar 4; then I take 1 part of this cream to 3 of the diluent, which gives a dilution of 4. By means of the Babcock test you can know the strength of your milk. You can take the milk to a creamery and have it tested by the Babcock method, or you can make the test in your office; we have it at the hospital. We need simplicity in the matter for home modification.

DR. HOLT.—Did I understand Dr. Chapin to say that it does not make any difference whether we are dealing with a 5, 4, or 3 per cent. milk, that the upper strata will contain the same amount of fat?

DR. CHAPIN.—No; that the ratio between the fats and proteids is about the same.

DR. HOLT.—I have come to practically the same conclusion as Dr. Chapin. The thought that interests me most is the short length of time necessary to allow milk to stand in order to get the cream in the upper part. People who have their own cows, for instance, and who are using the milk as it comes in fresh, how soon can they use it? I had a great many examinations made of different layers, taking four ounces at a time. I have found that after four hours' standing, if the milk were put into an ordinary milk bottle, practically all the cream would rise to the upper fourth. The proportions that Dr. Chapin found are practically the same as I found.

The main point in Dr. Westcott's paper, is the large number of children that do badly on high fats and low proteids. I believe if there is any one plan by which more children have been wrongly fed than any other, it is the facility with which fats can be increased, irrespective of proteids. Whereas a practically healthy child can take a high percentage of fat, in a child whose digestion is bad, you must cut down the fat a great deal, and much lower in summer than at any other time; it is probably the chief mistake we make. I think one reason that the mistake is so often made is the feeling that by increasing the fat we overcome early constipation. Now, while early constipation is no doubt an important matter, I am convinced that it is due to low total solids in the food and to nothing else. You must not expect two large yellow movements as in a child that is nursing from the breast. As long as the baby's bowels move every day I care not whether the size of the movement is a teaspoonful or ounces. If the baby is comfortable, constipation will disappear as the total solids are increased whereas if the physician feels that the constipation is abnormal and begins running up the fat rapidly he gets into trouble. I believe it is a mistake that so many make who think that the milk laboratory is of no value. It is so difficult to get these children straightened out when they have once gone wrong.

I think what Dr. Westcott says about the whey is a very good thing. We cannot predigest the fat and yet must keep it up.

DR. BUCKINGHAM.—I want to add my testimony to that of Dr. Holt and Dr. Westcott as to the harm done by too high a percentage of fat. It is the most common mistake that is made in feeding babies.

DR. MILLER.—I should like to endorse what Dr. Holt has just said about the amount of cream that rises from milk in a short time; the amount is quite uniform. The test for fat can be made by anyone in his office with the ordinary centrifugal machine. I should like also to express my appreciation of Dr. Chapin's dipper. I have found it a valuable and ready means of removing the cream, and it is far superior, because more easily worked, to the siphon. I have found that in treating the summer diarrheas of children the great difficulty in commencing milk has been the poor fat digestion of these infants, and, on this account, I sometimes use condensed milk because of its low percentage of fat. The indigestion of fat is not, in my experience, shown so often by vomiting, as it is by the presence of fat in the stools. For this reason I often find skimmed better than whole milk in summer diarrheas.

As to the use of whey, while I believe it is a very valuable food for sick infants, yet, it seems to me, as a method of feeding healthy babies, it adds to an already complicated process. The modification of milk has become so complicated that mothers refuse now to carry it out, and, it is a very difficult thing to make good whey. I have found, too, that in feeding babies on whey alone that they often pass a small amount of curd, for it is very difficult to get all the curd out of the whey. Then too, the whey must be pasteurized to destroy the rennin, another thing for the mother to do. When I first used whey, I often found the mothers showed me the bottle with the milk coagulated by the rennin of the whey. For these reasons, I think, whey can only be of service in infants who cannot manage ordinary milk, and is not necessary in normal children.

DR. COTTON.—When we write a prescription do we know what the patient gets? There is an interval between the writing of the prescription and administration of the dose. In preparing milk for babies each case must be made a problem itself and the doctor must do the work with his own hands, or his assistant must do it under his advice. We must ascertain what kind of milk we are using. There are hundreds of milk-men, delivering all kinds of milk. Every physician has a centrifuge in his office and ought to have a Babcock machine. When we get in the habit of doing this work, we are more ready to analyze breast milk and that is what we should be ready to do. When the milk disagrees we must analyze it and endeavor to remedy the defect.

We must try to bring the mother's milk into proper relation with the child's digestive powers before resorting to the use of other milk. It all means one thing, the thing that Holt and Rotch have been urging for years, and that is definite percentage feeding. I agree with all that has been said with regard to the loading of the child with fat.

DR. KOPLIK.—Meigs of Philadelphia, in his cream mixture, advised that the milk be put aside for three hours in a cold place. If my patients are so situated that they cannot obtain bottle milk, I advise them to put the milk aside for three hours and then take off the cream.

DR. TOWNSEND.—I agree with Dr. Chapin that the greatest simplicity in the modification of milk is desirable, not only for the physician who may not be a mathematician and cannot carry the formulæ in his head, but for the nurse, and most of all for the baby. I have given up the use of the siphon which is in some hands a dangerous instrument. I have not used Dr. Chapin's dipper, which I have no doubt is a very good thing, but I have the top milk simply poured off. I have found by analyses that the upper quarter poured off contains about 10 per cent. of fat, and the upper half 7½ per cent. of fat.

As to the whey, it seems to me that the manipulations necessary in making it are undesirable. Where it is necessary to use low percentages of proteids and fats I have found the method of adding white of egg a very good thing. It adds to the nourishment of the mixture and puts little work upon the digestion of the baby. Of course as we raise the percentage of milk albuminoids we can gradually dispense with the egg.

I want to speak of a case that illustrates the fact that babies are not machines and may not follow any rules. The patient a seven months old, healthy child, was taking top milk and barley water, when it began to spit up a little of its food after each feeding, but was gaining weight and the bowels were all right. I modified the food in various ways, reducing the fats, but without any effect. At the suggestion of the mother, who said her former child did the same thing, I put the baby on pure milk, when it stopped the spitting up at once. This simply shows that we cannot always reason about babies theoretically, and we must modify our food to the baby, and not expect the baby to take a certain modification because it is theoretically correct.

DR. EDSALL.—In connection with the class of cases reported by Dr. Westcott, in which there is disturbance of the stomach, rather than of the intestine, from undue quantities of fats, I think that it is worth while to direct attention to the recent work of Vollhard, which has shown that there is a fat-splitting ferment in the gastric secretion. He worked entirely on adults;

and his studies show that the ferment acts only on fats that are well emulsified, and only in the absence of any notable amount of hydrochloric acid. Hence, it is evident that the ferment is not of much importance in the adult. But the young child takes all its fats in a well-emulsified form; and the acidity of its gastric contents is, at most, low even in health. It seemed possible to me, therefore, that this ferment is chiefly useful in the child; and that in the adult it is only a remnant of a function active in childhood. I have satisfied myself, at any rate, that the ferment is present in the child, and that in some instances it is very active. I have attempted to provide a method for determining its presence and activity, clinically. Vollhard's method takes several days to carry out, the main difficulty being in drying the mixture in which the ferment has been acting, so that it can be extracted with ether. I have used acetone instead of ether, because it has the property of dissolving fats in watery solution. With this procedure, I can carry out the examination in a very short time; and, if it proves to be accurate, the method will be suitable for clinical use, and may be of interest in helping to determine a cause for disturbances such as those which Dr. Westcott mentions, and also possibly in influencing diet.

In connection with the figures for milk-protein in Dr. Chapin's table, I would say that I do not think that we should make up our milk-mixtures on the basis of such figures. If I remember correctly, they show, in all but one instance, less than 3 per cent. of protein. This certainly does not fairly represent the milk that we ordinarily use, more particularly in the better class of patients. It has been thoroughly demonstrated that such milk usually runs higher, and I have myself made 150 or 200 Kjeldahl nitrogen estimations of milk, and in only three or four instances, I think, did I find the protein running below 3 per cent. In almost all cases, it was as much as 3.5 per cent. and frequently it was as high as 4 per cent. the average being in the neighborhood of 3.75 per cent. This was good, mixed dairy milk; and I think that these figures are much closer to those of the milk that we ordinarily use. I also do not agree that the fats in dairy milk are likely to run pretty evenly, if milk from the same source is used. I have seen fats that were estimated daily, in milk from the same dairy, vary from 3 to 5½ per cent., from one day to the next, and decided variations are common. Our milk mixtures are made from day to day, and are, therefore, subject to such variations.

DR. FREEMAN.—I have been rather surprised that the one really definite method we have of getting cream has not been mentioned at all, and that is the method of ordering definite percentage creams from the laboratories, and I think where we have the laboratories we are foolish not to use them, so that we

can know just what we are getting. I think in the country a good way is to have milk from the evening milking left over night and then use the skimmed cream, which is 16 per cent., and dilute to get the desired percentage. There is no objection to having it kept twelve hours, if kept in a not very warm place, for in clean milk the bacteria usually diminish during that period. I noted some years ago that in several observations there was a constant diminution in the number of bacteria in milk for twenty-four hours after milking. Fokker in Germany noticed the same fact in eleven observations. A thorough piece of work on this subject has recently been carried out at Cornell University in which the same facts are observed.

DR. WENTWORTH.—In order to escape the charge of plagiarism, I should like to say that I have recently sent an article to the press in which I made practically the same statements that have just been made by Drs. Chapin and Holt, namely, that to insure even approximate accuracy in the modification of milk, it is necessary to determine the percentage of fat in the cream that is used for modification; and that too high a percentage of fat in milk mixtures, frequently causes serious digestive disturbances in infants.

I prefer the Chapin dipper to the siphon for removing cream from milk.

With reference to Dr. Westcott's paper I must say that the results which I have obtained from the analysis of a considerable number of "milk-modifications" have convinced me that it is impossible to procure accurate modifications of milk unless complete analyses are made of the milk and cream used for modifying purposes. I should like to ask Dr. Westcott whether the percentages of fat, sugar and proteids were determined in the mixtures that he has recommended?

Dr. Westcott admits that the fractional subdivision of the ingredients in milk-mixtures is only needed for infants that are unable to assimilate the ordinary milk-mixtures. My experience has convinced me that the best thing to do is to procure wet nurses for these infants. It is of the utmost importance in these cases to avoid as much as possible, any further loss of time.

DR. GRIFFITH.—I wish only to call the attention of the Society to something that all of us know but are apt to forget, namely, that the use of whey dates back many years. The results of my own experience with it have been excellent. I have repeatedly found a mixture of whey and egg albumen agree and nourish a child when I failed with stronger and fatter foods.

DR. WESTCOTT.—The practical scope of my paper was a limited one and applied only to children that I denominated

difficult cases,—that is, cases of chronic gastric catarrh, or of chronic high intestinal catarrh.

I consider Dr. Chapin's cream dipper a very useful device, especially in summer, when both centrifugal and gravity creams are sources of danger. As to variations in the percentage of fat in gravity cream when obtained from milk from the same dairy, and with the same routine method from day to day, I have rarely observed disturbances of digestion as long as the same theoretical formula was maintained. On the contrary, when the theoretical formula has been changed often by so little as would be produced by one or two teaspoonfuls' increase of whole milk, disturbances have been found frequently and immediately to occur. This seems to me to indicate that for practical purposes, in home modification at least, the basal percentages in the cream and milk used for feeding any individual case may be assumed as constant, and that theoretical variations in formula obtained by calculation may be considered as actual and relatively corresponding. In support of this it may be noted that a difference of say 0.50 per cent. in the fat of the basal milk would be represented by a variation of only 0.125 per cent. in a one in four dilution, or 0.10 per cent. in a one in five dilution, and that these variations are more apt to be on the side of deficiency, since the constants used in calculating formulæ are, if anything, maximum values. The error is, therefore, likely to be on the safer side.

I agree with Dr. Chapin as to the principle of working backward, and very often use this method of determining percentages conditioned by the older method of combination. All the percentages given in my paper were worked out in that way.

Dr. Holt has spoken about constipation which I agree with him is due principally to the low total solids.

It is an interesting fact, and as I believe not generally understood, that in the chronic catarrhal cases, gradually increasing constipation is a forerunner of an acute exacerbation of the disease. These annoying interruptions may occur at first as frequently as once a week, but as the case goes on to recovery they happen less frequently and with constantly lessening severity. The mother soon learns to anticipate a fresh exacerbation by the fact that for several days beforehand the bowels become more sluggish and difficult to move.

As to Dr. Miller's remark about using whey for feeding healthy babies: I did not mean that at all. It is only where the casein proteid that can be digested is too low to supply the requisite amount of nourishment that I advocate whey feeding. Generally speaking, a child four or five months old is not getting enough proteid until the percentage of this element reaches 1.50. The case I referred to I did not bring forward as a prize case, but it was a very trying case, and the physical condition of

the child is now exceedingly satisfactory. I have on the same chart the curve of another child (marked case II) fed on the same sort of mixture, and the rapidity of weight gained is distinctly shown by the rapid rise of the line. (See chart).

Dr. Miller referred to the difficulty of making whey. This has not proved a formidable task to many mothers under my direction, who with little practice have become expert in making it. What has seemed to us the best method of curding is by the use of the ordinary junket tablet of the household, about one and a half tablets to a quart of milk. After the whey is strained it must be raised to a temperature of 150°, as Dr. Miller states, to destroy the excess of rennin, which otherwise would cause coagulation of fresh milk mixed with the whey. This preliminary heating is not necessary if partially peptonized milk, instead of fresh milk is to be used in the combination. It is perhaps safer to do it, however, for its pasteurizing effect.

DR. CHAPIN.—As to Dr. Edsall's statement about low proteids, this study was not intended to show low proteids in any class of milk, but how the soluble proteids varied. It is very difficult to establish any ratio between the casein and soluble proteids.

With reference to the use of the laboratory, the majority of students we see do not live within hundreds of miles of laboratories and this simple method will give almost as accurate a cream as the laboratory. What we want are methods that can always be applied.

SEPTIC ENDOCARDITIS.

BY SAMUEL S. ADAMS, A.M., M.D.,

Washington, D. C.

The interest manifested by my colleagues of the Children's Hospital, Washington, D. C., in the recovery of a case of septic endocarditis under my care, and the recital of other recoveries from this disease by members of the Association of American Physicians, have induced me to collate the cases in children fourteen years of age and under which have resulted in cure, as well as to review the literature of endocarditis in its relation to infection as the causative element. The difficulties encountered in this attempt were often embarrassing, and at times appalling, because (1) the subhead "Endocarditis," as indexed at the Library of the Surgeon-General's Office, is lost under the headings "Heart (inflammation of)," "Heart (disease in children)," etc.; (2) a number of the cases here collated were not indexed, and in many instances were traced from allusions to them, found by collateral reading; and (3) owing to the confusion in the classification of the varieties of endocarditis, the greatest liberality being shown in the descriptive adjective; on the one hand the terms "simple" or "benign," and on the other "ulcerative," "malignant," "infective," "diphtheritic," "septic," "fungating," and "mycotic," being subject only to the license of the author or reporter.

The effort has been made to collate only such cases as may be considered infective from the specific statement of the author, or from the characteristic clinical picture by which the disease is more frequently diagnosticated than from either bacteriologic or post mortem findings. It is not claimed that every published case of cure is included in this list, for in all probability there are some recorded in text books or incidentally mentioned in other works which have escaped us, but it is certain that all indexed cases, as well as those which were accidentally discovered, are here reported.

Unless we attribute the small number of cases reported to failures in diagnosis, septic endocarditis in children resulting in recovery may be considered to be a rare affection. Donkin says it is not frequent in childhood, and has no clinical peculiarity at this

period. He saw only 3 probable cases, which were suspected during life, only 1 having been submitted to post mortem examination, which alone gives positive evidence of this disease. It is sometimes found post mortem where it is not suspected, but it is still more often suspected where it is not found.

Gorwitz, on the other hand, draws the following conclusions: (1) By the side of the classic, infectious endocarditis, we place, in the cases described, a form as yet little known. (2) This form is rather peculiar to infants. (3) The beginning is insidious, the progress slow, the heart is little affected, as also the other organs. (4) The infection dominates the scene, leaving in the second plane the circulatory apparatus. (5) The prognosis, while grave, is not (*necessarily*) fatal; it is subordinate to the degree of infection. (6) The invalid may recover, but the affection leaves, as a trace of its passage, an organic lesion of the heart.

Osler says that in 209 cases of endocarditis which he has studied, there were but 3 under ten years of age. Whittaker says: "It is rare in infancy, but begins to show itself with the occurrence of various infections." The records of the Children's Hospital at Melbourne furnish but 1 case in ten years. Davies finds but 1 case similar to his own in the Transactions of the Pathological Society (London); it is that of a girl one year old, under the care of Dr. Sharkey. This case I have been unable to find elsewhere.

The earliest case, that recorded by Kirkes, who first recognized and described the disease, was in a boy aged fourteen years. Only a single case appears in the records of the London Hospital for Sick Children during the two decades ending in 1890; and my case is the only one treated in the Children's Hospital, D. C., since its organization in 1870.

Kanthack and Tickell have studied 84 cases in St. Bartholomew's Hospital from January, 1890, to March, 1897. The following were found in children: By age, 1 to 5 years, 1 male, no female; 5 to 10 years, 2 males, 2 females; 10 to 15 years, 2 males, 4 females. Of these 84 cases, 60.7 per cent. were males, 39.29 per cent. females. 62.7 per cent. of males between twenty and forty years; 51.5 per cent. of females between ten and thirty years; 33.3 per cent. of females between fifteen and twenty-five years.*

* This note is added lest the relative predominance of females in list lead to erroneous conclusions.

In connection with the statistics given by Kanthack and Tickell, those of Thiele are interesting. Between 1873 and 1887, 4,979 necropsies were made in the Pathologic Institute at Kiel. Of these endocarditis was found in 373 cases, 168 (45 per cent.) in females, 209 (56 per cent.) in males, and the sex not stated in 1. Under 1 year, 5 males, 6 females, total, 11. One to 9 years, 10 males, 8 females, total, 18. Ten to 19 years, 14 males, 12 females, total, 26.

Fresh hemorrhages, old hemorrhages, infarcts, and embolic abscesses were found in the brain, retina, lungs, liver, spleen, and kidneys.

While it is generally admitted that septic endocarditis is a specific affection, nevertheless there have been many pathologists, bacteriologists, and clinicians unwilling to accept it as such. Lazarus-Barlow contends that, in spite of the fact that physicians recognize two varieties of acute endocarditis (simple and malignant), and that they differ in their clinical pictures, the difference between simple and septic endocarditis is one of degree, dependent upon the virulence of the microorganisms or their toxins. He admits that microorganisms are almost invariably found in the vegetations on the valves in infective endocarditis, but asserts that they are frequently found in the so-called simple variety, and that the same microorganisms are present in both conditions. According to his interpretation, the microorganisms act as irritants, producing not only inflammation in simple, but inflammation plus necrosis in septic, endocarditis. His explanation of the difference in the pathological processes produced by the emboli which are broken off from the vegetations and carried in the blood-currents is that such emboli are aseptic in simple, and septic in infective, endocarditis. The effects also of the two varieties upon the heart itself are merely differences in degree; both forms produce changes in the myocardium, though the ulcerative, from the effects of its toxins, produces greater impairment of function of both heart muscle and valve.

Weichselbaum claims that there is no essential difference, either histologically or bacteriologically, between the two forms of endocarditis; that the same species of bacteria are found in both; and that in those cases in which bacteria have not been found they have either escaped detection or have died.

The trend of opinion to-day seems to accord with the views

of Lazarus-Barlow and Weichselbaum that the two conditions differ in degree but not in kind; and one author suggests that the burden of proof falls upon him who would present a case of noninfective endocarditis.

Rosenbach, in 1878, induced acute endocarditis, experimentally, in animals by injuring the heart valves with a sound passed into the carotid; and, in 1885, Wyssokowitsch and Orth induced it by injecting pyogenic organisms into the circulation after the heart valves had been injured by Rosenbach's method.

A study of the experimental work of Netter, Ribbert, Fränkel and Sänger, Prudden, Weichselbaum, Stern and Hirschler, Viti, and others shows that true endocarditis can be produced in animals by injecting in the veins any one of the numerous microorganisms, provided the heart valves are diseased, although Netter and Ribbert think a previous lesion is not essential; and Dreschfeld and others have succeeded in reproducing the disease in animals without previously injuring the valves.

According to Wright and Stokes: "The first extensive study, however, of the etiology of acute endocarditis in man is that of Fränkel and Sänger, published in 1886. They studied 11 cases of verrucose and 1 of ulcerative endocarditis, and in 9 of them found bacteria in the cardiac lesions, the staphylococcus pyogenes aureus being present in 6." Netter found the micrococcus lanceolatus in 9 cases of acute endocarditis associated with croupous pneumonia. Of the 29 cases of Weichselbaum, the micrococcus lanceolatus was found in 7, the streptococcus in 6, the staphylococcus pyogenes aureus in 2; and in 6, various unusual bacteria; while in 8 cases no microorganisms were found. In 10 cases, Wright and Stokes examined the valvular vegetations and ulcerated tissues of the heart and found the micrococcus lanceolatus in 7, 1 being accompanied by the bacillus diphtheriæ; the streptococcus in 1; streptococcus and staphylococcus in 1; and unknown in 1.

Dessy found in 36 cases of endocarditis (3 being ulcerative and showing pneumococci) bacteria in 34; viz.; pneumococcus in 13; streptococcus in 12; staphylococcus in 3, and other species in 6. He considers the pneumococcus and streptococcus, either alone or associated with other forms, the cause. Lancereaux says that when infective endocarditis is found in connection with malarial poisoning, a frequent occurrence in Africa, its seat of predilection is in the aortic valves. Dessy informs us that the pneumococcus

selects the aortic valve, and the streptococcus the mitral. Again, Raue explains "the almost invariable occurrence of endocarditis upon the left side of the heart, during extrauterine life, and a similar selective affinity for the right heart in fetal endocarditis," upon the ground that "endocarditis is an infectious condition" and "the germs which excite inflammatory reaction in the endocardium can thrive only in a medium of arterial (*i.e.*, freshly oxygenated) blood"—an idea perhaps indicated by Giraudi.

Dreschfeld summarizes the results obtained by different observers as follows:

1. That microbes were found in nearly all cases of infective endocarditis, regardless of ulceration.

2. That most observers found but one organism in their cases; a few, however, found two or more.

3. That the same organism was not found in all. In some the organism found was also met with in other infectious diseases; and in others the organism had not been found in other diseases.

4. In most cases, the organism found in the diseased valve also invaded the infarcts and metastatic abscesses.

5. The streptococcus, the staphylococcus, and the pneumococcus are the organisms most frequently found.

D'Astros comments on the difficulty of demonstrating the several steps of these infections during life; Achalme, for instance, in a case of endocarditis, complicating erysipelas, having failed to find the streptococcus during life.

The disease may be considered rarely primary. Osler states that it is engrafted upon a preexisting valvular lesion in 75 per cent. of cases; hence the microorganism invades the broken endocardium.

Dreschfeld says we may distinguish the following types of infectious endocarditis: (*a*) primary; (*b*) as a complication of septic disease; (*c*) as a complication of pneumonia or meningitis, and due to the diplococcus pneumoniæ; (*d*) as a mixed infection, due to septic organisms secondary to the acute infectious fevers, or secondary to rheumatic endocarditis or sclerotic condition of the valves.

Lazarus-Barlow says in many cases of pyemia a primary focus of suppuration is found, but this is not invariably the case, to account for the metastatic abscesses. In such cases cardiac symptoms

develop and the necropsy reveals ulcerations on the valves. He admits in such cases the lesion in the endocardium as the immediate antecedent cause of the abscesses, because the pyogenetic organisms have been found there. "Where this is the case, association of the secondary abscess with embolism of particles of the septic vegetations on the valves is easy, especially in view of the fact that in these cases the metastatic abscesses are most commonly found in the spleen and kidneys, regions in which simple embolism and infarction are especially common in valvular disease of the ordinary kind." It is not easy to explain the valvular disease, which, in a majority of cases, antedates the ulcerative process. The microorganisms must gain access to the blood in order to produce their harmful effects. In all cases in which the microorganism is localized, and there are associated with it valve lesions and metastatic abscesses, the relation between cause and effect is apparent. The blood is invaded from a primary focus situated in the tissues. He assumes, therefore, the preexistence of a primary superficial focus, from which emanates the septic material in so-called primary ulcerative endocarditis. Micrococci may set up an ulcerative process in a simple valve lesion. It is probable that a valve affected with a chronic lesion may be unable to resist the invasion of pyogenetic microorganisms circulating in the blood. It is not necessary that such organisms should gain access to the blood immediately preceding the manifestation of the endocarditis. We believe that various forms of pyogenetic bacteria may remain latent in the body for a long time, and so that form of bacteria which is to produce the ulceration on the endocardium may have entered the body and have been stored in some tissue capable of resisting its pathogenic influence. At last some condition arises which leaves it free to gain access to the general circulation, when it attacks the diseased valves with great virulence.

This view is supported by the conclusions of Gorwitz, already given, as also by Anders, who says: "In purely septic diseases ulcerative endocarditis forms but a part of the serious general condition. Here the cardiac element serves to facilitate the generation and rapid diffusion of the poison; and since the latter is prone to attack the valve segments, the morbid lesions within the heart not rarely constitute a chief pathologic factor in septicopyemia."

Everything except the main points in the history of my case has been eliminated.

ADAMS' CASE. Female, six years. Admitted Dec. 14, 1899.

FAMILY HISTORY.—Negative. Always delicate. At four years of age had measles; good recovery. Health good until August, 1899, when she was treated in this hospital for remittent fever, estivoautumnal type; not severe, but weakening. No cardiac lesion. Discharged, cured, September.

December 1st, Readmitted.—Severe chill, fever, sweat, intense headache. Next day another chill. Estivoautumnal parasite found. Quinin sulphate given.

December 9th.—Tired, quiet all day.

December 15th.—Temperature 103.4°. Dyspnea, brief unconsciousness, continuous high temperature, 104°; pain in abdomen and precordium. Well-nourished, very anemic, distressed look. Respiration rapid, not embarrassed. Chest well formed, equal expansion. Slightly impaired percussion resonance at right scapular angle. Loud bronchovesicular breathing at left apex; almost bronchial at right scapular angle; many râles, moist and crepitant. Breath offensive, appetite poor, tongue coated, bowels constipated. Throat, abdomen, liver, spleen—negative. Pulse full and bounding, impulse heaving. Marked apical thrill; beat diffused over space one inch in diameter; area of dullness but slightly increased. Harsh, strong, presystolic murmur at apex, not transmitted. Second sounds much accentuated, especially pulmonic. Urine diminished, thick, cloudy; many amorphous urates; no albumin nor casts. No pain. Sleeps poorly. Interrogated: "Feels all right." (See cut.)

December 16th.—Temperature range 101° to 103°. Slept fairly. No urine at night. Pulse rapid and strong. Heart murmur slightly changed, double at apex. Small area dullness, left scapular angle, with bronchial breathing.

December 18th.—Temperature the same, irregular, with evening rises. Presystolic murmur and thrill have disappeared. Soft, blowing, systolic murmur transmitted to axilla; other valve sounds clear and distinct; pulmonic second sound still markedly accentuated.

December 20th.—Same condition, with slight to-and-fro friction at base of heart; action tumultuous and plunging; apex beat much diffused; dullness much increased.

LARGE CIRCLE: AREA OF RELATIVE DULLNESS OF HEART. SMALL CIRCLE: DIFFUSION OF APEX BEAT. STARS: AREA OF ABSOLUTE HEART DULLNESS. A: AREA OF DULLNESS AND BRONCHIAL BREATHING.

From Jacobi Festschrift, Courtesy of G. P. Putnam's Sons.

December 22d.—Examination by Dr. Wm. B. French for plasmodium malariæ negative. Slept poorly past two nights, vomiting nourishment several times. Stopped quinin. Pulse weak, rapid, and at times irregular. Given tincture of digitalis. Voids only eight ounces urine in twenty-four hours.

December 26th.—Temperature normal, respiration rapid and labored. Sleeps better; does not vomit.

December 30th.—Temperature 1 P.M., 102°; respiration as before. Pulse full and bounding; heart-sounds muffled, with marked double murmur at apex, change coincident with rise of temperature.

January 1, 1900.—Temperature intermittent, 98.8° to 103.4°. Oral mucous membrane shows small purplish spots. Respiration very labored. Pulse weak and irregular at night, full and bounding during the day. Retains all nourishment. Splashing sound at base, marked systolic murmur at apex transmitted to axilla, second pulmonic markedly accentuated; impulse heaving and diffuse. Child sleeps with eyes half closed; breathes altogether through mouth; rests well; no pain.

January 3d.—Single systolic apical murmur, transmitted to axilla. Perspires freely about head and neck, has had several drenching sweats in the past twenty-four hours. Spots disappeared from mouth. Retains nourishment. Bowels constipated.

January 5th.—Has vomited a great deal, clear green fluid, streaked with blood. Refuses nourishment. Does not sleep at night. Cries a great deal, but no pain complained of. Temperature dropped by crisis, being 99.8°.

January 7th.—Nourishment retained. Urine still diminished in quantity, quality the same. Face slightly swollen. Pulse full but compressible, at times weak and irregular. Sleeps much better.

January 8th.—Temperature commenced to rise.

January 9th.—101° to 102°; other conditions unchanged.

January 11th.—Temperature still elevated. Pulse and respiration rapid and full. Cried several times during night; no pain complained of. Murmur same, loud, blowing; obscure valve sounds; splashing audible at base.

January 13th.—Sleeps poorly, seems gradually weaker. Cough frequent. Temperature about 103°, with frequent excursions to 104°. Urine still diminished; perspires freely; marked

odor of urine. Numerous moist râles, large and small; diminished breath-sounds at bases, rough elsewhere.

January 15th.—Yesterday, pain in cardiac region increased on coughing. Vomits nourishment frequently. Otherwise the same.

January 17th.—Restless at night, crying and picking at face and bedclothes; complains bitterly of pain, stabbing in character, in region of heart; tenderness on pressure. Cough frequent and hard. Nourishment retained. Stools involuntary, large and loose.

January 19th.—Slept better, less pain. Temperature dropped to 100°. Cough frequent. Urine diminished.

January 23d.—Stools involuntary, large, yellow, undigested. Temperature intermittent, 100° to 103°, rise in evening. Cough frequent and hard, no expectoration. Splashing not heard; sounds still obscure; churning sound; impulse forceful and heaving; apex beat much diffused. Lungs same.

January 25th.—Culture from vein in arm inoculated upon three blood serum slants. After twenty-four hours' incubation, the culture shows numerous streptococci and another unrecognized bacillus. (Dr. French.)

January 26th.—4,500,000 red blood corpuscles per cm. Hemoglobin 62.5 per cent. Leukocytosis. (Dr. Thomas.)

January 27th.—Photographed; cardiac area and apex impulse outlined with ink. Sounds changing somewhat; impulse not so heaving; churning not so audible; valve sounds more distinct; murmur not so prominent, nor does it obscure sounds so much. Temperature continues about 100° to 101°.

January 31st.—Temperature made another rise this A.M., but dropped quickly to 99.6°. No pain. No other change noted.

February 10th.—Cardiac area much increased; impulse heaving, and widely diffused, noted over whole cardiac area. First sound obscured. Murmur loudest at apex, traced to left scapular angle. Systolic splashing and churning not heard; slight rubbing at base. Sleeps well and takes all nourishment.

February 20th.—Condition of heart unchanged. Has whooping-cough, ten to fifteen paroxysms daily which greatly exhaust her.

November 1, 1902.—This child remained in the institution until the summer of 1900. Soon after her recovery from whoop-

ing-cough she had a severe attack of measles, complicated by croupous pneumonia; she had only reached convalescence when chicken-pox of a severe type jeopardized her life. She slowly gained strength and weight; the heart's action steadied, and she was removed to a western state about the middle of July. I have heard from her but owing to the remoteness of her residence from a town it has been impossible to have a physical examination made. Her grandmother declares that she is now a robust, romping schoolgirl; that she is a bright pupil, and that no one would ever imagine from her present appearance that her life was ever in danger.*

Of 47 cases under fourteen years collated, there were only four recoveries, including my own case. The histories in brief of the three are as follows:

CASE I.—Gorwitz (Mlle. C. R.), Paris Thesis, Abst. *La Med. Infantile,* 1894, I., 475.

Male, eleven years. Rheumatism. Infectious endocarditis. Recovery in two months with persistent cardiac lesion.

CASE II.—Sainsbury. (H.). *Lancet,* London, 1896, II., 1079.

Male, thirteen years. Thrill and double apical murmur. (June 22d.) T. 103.2° F.; (24th) violently delirious. T. 100°-101°; no streptococci in blood. (25th) Rusty sputum, no rigor. (26th) Symmetrical erythema, general, five days. (July 1st) Streptococci in blood. (9th) General improvement. (20th to 24th) Rash returned, cardiac dullness increased. (28th) T. 104.4°, no rigor, but vomiting, cough, bloody sputum. (August 5th) Worse; sputum blood-streaked and clotted; thought spleen palpable. (17th) 20 c.c. antistreptococcic serum, T. 99.8°; no local nor general reaction. (18th and 20th) 10 c.c. serum; improvement; skin peeled and cleared. (22d and 25th) 10 c.c. serum. (26th) Rash and much itching. No streptococci. (September 1st) 10 c.c. serum, slight reaction, local and general. T. 102°, patient seemed ill, pain at puncture, twenty-four hours; recovery.

CASE III.—D'Astros. (Leon), *Rev. Mens. des Mal. de l'Enfance,* Paris, 1898, XVI., 601.

Male, seven years. Erysipelas. Auscultation, nothing abnormal at first. T. reached 106.8° F., pulse 142. Two injections, 10 c.c. each. Recovery. Persistent mitral lesion.

* This paper has been withheld in the hope that the present condition might be annexed, but waiting seems in vain.

DISCUSSION.

DR. CRANDALL.—I think our ideas as to septic endocarditis would be clearer if we got rid of some of the old terms, particularly that of "ulcerative endocarditis" and understood that endocarditis is secondary to some condition that has preceded it. The endocarditis of rheumatism is caused by the rheumatic poison, and we look upon it as a complication of the original disease, and so with septic endocarditis. If it occurs with pyemia we recognize it as secondary, but when the septic conditions are less clear then it does not seem so plain. Still it is probably always the case that there is some general septic condition preceding it. I saw two weeks ago two specimens of hearts one of which had occurred in the course of rheumatism, and the other in the course of a septic disease. Yet everyone who saw the two specimens selected the first as the septic one. It is not always possible to determine from post mortem appearances what was the cause of the endocarditis.

DR. WILSON.—I think our conceptions as to certain forms of disease are influenced by the terminology used. In regard to endocarditis, the classification may be bacteriological, pathological or clinical. Often the bacteriology remains obscure and while the physical signs and existing history may justify conclusions as to gross pathological changes, in many cases both the bacteriological and pathological changes can only be inferred.

On the other hand, clinically, the different forms are rather more distinct and it seems to me that it is best to adhere to a nomenclature more distinctly clinical. We have to deal with forms properly called benign, many of them so benign that in the absence of physical examination their existence would not be suspected. On the other hand we have to deal with cases of endocarditis obviously grossly malignant. Yet from a bacteriological and pathological standpoint there is no distinct line of demarcation between these two extremes. At one end of the series we have, both bacteriologically and pathologically, the benign cases, at the other the malignant and midway between these cases, in which it would be a little difficult from the clinical phenomena to say whether they should be regarded as benign with aggravated symptoms, or malignant with mild symptoms.

So much for the physiological standpoint. Speaking now from the standpoint of the teacher, with reference to cases presenting symptoms of the malignant type the symptom complex which goes to make up the typical, well marked forms is characterized by recurrent chills, irregular high temperature, profuse sweating and rapid wasting, with the development of physical signs that are characteristic of coarse changes in the endocardium, and particularly in the valvular endocardium, and by the frequent occurrence of embolism in various parts of the body. There are cases that arise from septic processes in distant parts of the body, necro-

tic processes, septicemia, etc., which usually terminate fatally, but occasionally in recovery, as for example, in puerperal sepsis. Then there is a group of cases in which it is not uncommon for all these symptoms to arise and the patient recover. There are especially cases of recurrent endocarditis in old valvular disease originally of rheumatic origin. I have had under observation in the last five weeks a case of that kind which is now recovering. The severe symptoms have passed away, although for a week or ten days the outcome was very uncertain. The patient is now sixteen years old, and has had chronic valvular disease involving both mitral and aortic valves, three or four benign attacks, but the attack through which the patient is just passing was malignant with all the phenomena of that type emphasized, not excepting embolism and monoplegia of the right arm.

DR. MILLER.—I am not familiar with septic endocarditis in childhood, but have seen the same condition apparently get well in adults. In very young infants septic endocarditis, I should think, would be rather frequent, as septic conditions are very common in the new born. I remember treating a case of chronic valvular disease of the heart in a patient at the age of two years, the initial lesion of which must have occurred many months earlier. Such cases are not uncommon, and are due to attacks of endocarditis dating from early infancy.

DR. JACOBI.—Such cases are caused by either bacteria or toxins. Bacterial deposits on the valves, usually below the valves, generally near their insertion, are those which would probably be called malignant, for from them emboli will be taken up and septicopyemia produced. The second class are those cases which are not the result of bacteria, but of toxins. They are those which are apt to get better. The toxin will be eliminated after doing a great deal of damage, but a toxic carditis may get entirely well by this elimination, as for instance in influenza and typhoid. That is an occurrence which is quite frequent. Six or eight months may suffice to restore the organ to its normal, healthy condition.

Then there is another difficulty. Is it always an endocarditis with which we have to deal, or is it not rather often a myocarditis? The murmur in myocarditis will often give us the impression of endocarditis. There need not be bacteria in the case at all. In those cases where we have bacterial deposits on the valves I do not think we can safely expect to have recovery take place, but in toxic endocarditis very often. The principle difficulty is that we make the diagnosis of endocarditis when we have myocarditis, for the murmurs may be exactly the same. If there is a point of diagnostic importance it is this, that in chronic cases there is absence of dilatation and hypertrophy in myocarditis, but the presence of hypertrophy where we have to deal with valvular disease. We

should make this distinction first, whether myocarditis or endocarditis, and, secondly, whether bacterial or toxic. We cannot prove when an alleged endocarditis gets well that it was not a myocarditis we had to deal with. I think that most of the cases we find getting well have been myocarditis.

DR. SEIBERT.—I would like to ask the bacteriologists present if I am right as to whether Wasserman did not publish, the case of a young girl about a year and a half ago, who died of endocarditis within three days of the beginning of the attack, where he succeeded, for the first time, in taking bacteria from the endocardium which lived in the culture media he selected, and from which cultures he succeeded in artificially producing acute articular rheumatism in animals? The reason he had not formerly succeeded in cultivating this germ was that the culture media that he had used before had not a sufficient degree of alkalinity, thereby proving that the doctrine we were taught, to render the blood of rheumatic patients alkaline, was aiding the bacteria in maintaining life in the blood circulation.

The question in this case of Dr. Adams is what is septic endocarditis and what is rheumatic endocarditis? I believe the only distinction we can make is that made by bacteriologists and from them I am quite sure we would have malignant rheumatic endocarditis as well as malignant purulent endocarditis.

DR. ADAMS.—From the physiological standpoint I agree with Dr. Wilson. I think a great deal of confusion as to terms has arisen. Somebody says it is malignant and we accept it as such.

Now this child was treated in the hospital in August for the estivoautumnal variety of malarial fever by quinin and was apparently cured. Dr. Acker was attending to my service and he examined the child carefully and his notes show that there was no evidence whatever of any heart disease. She was readmitted in December, again carefully examined, and still no organic disease of the heart detected. She was then treated, before the blood examination was made, for the estivoautumnal variety of malaria, supposing it was a recurrence. She did not respond to the quinin this time, however, and then the bacteriologist reported that no organisms were found in the blood. The quinin was discontinued, the septic condition became more and more manifest and the murmurs made their appearance. With the recurrence of the chills and sweating we came to the conclusion this was a septic heart. I have had one case in an adult (the man died); and the picture was exactly like this child. Dr. Reed made the cultures from the heart which showed that it was what we recognize as septic endocarditis. I would not have presented the case but for the fact that at the Association of American Physicians Dr. Herrick reported some of these cases and Dr. Koplik asked me to report this one to our Society.

THE EFFECTS OF TIGHT DIAPERS.

BY A. C. COTTON, M.D.,
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The main object of this paper is to deal with some of the disadvantages of the clothing of infants and to advance a few suggestions as to the best means of obviating them. One of the first apparent disadvantages is the discomfort of clothes to the infant and the annoyance incident to its manipulation. After food and warmth, the prime essential for the infant's welfare is rest. Clothing that requires frequent or prolonged handling, which causes discomfort from constriction or pressure, or from skin irritation, which induces fatigue by weight or constraint, or causes overheating or exhaustion, is familiar to us all. It does not allow the infant to rest.

Clothing may interfere with functions, as of respiration by compression of the thorax; of circulation, by constriction of the blood vessels and by restrained muscular movements; of digestion, by interfering with gastric and enteric peristalsis with resulting fermentation, constipation and their train of evils; of elimination, by deficient skin ventilation; of voluntary muscular action, by restraint which prevents absolute freedom of the extremities.

There is no doubt that improper clothing not only may interfere with growth from impairment of function, as above claimed, but it may also cause actual deformities from undue pressure upon plastic tissues in their formative states.

The misshapen head, due to the infant's inability to change position because of its clothing, is not infrequently seen. The constricted chest with undeveloped lungs may not be due solely to rachitis, but to compression. Constriction of the abdomen may favor hernia, permanent gastric and intestinal dilatation, deformed liver with its chondral grooves and atony of all the muscular structures.

That a diaper of unyielding material pinned firmly around the hips of a young infant will compress the pelvis, reducing the diameters is susceptible of demonstration, as may be seen in the accompanying skiagrams, A1 and A2, made from the same subject (See Figs. A1 and A2.)

FIG. A1.—NORMAL INFANT PELVIS.

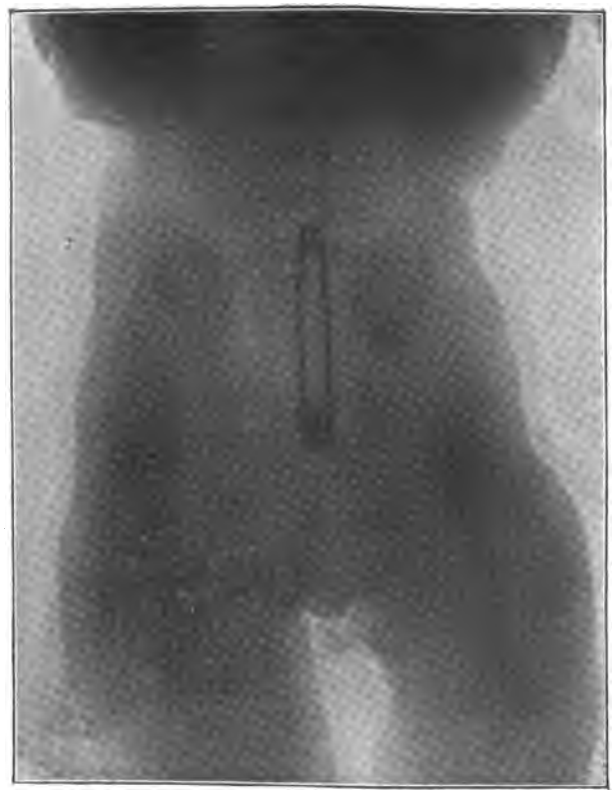

FIG. A2.—SAME PATIENT AS A1; SHOWING EFFECT OF TIGHT DIAPER ON PELVIS.

The effect of such compression applied continuously throughout the first year of life, it seems reasonable to assume, would be a retardation of pelvic development, if it does not cause a disproportion of the relative dimensions. No one doubts that the thorax may be constricted in growth by prolonged compression. The effects of this compression are also shown by the x-ray in skiagrams B1 and B2. (See Figs. B1 and B2.)

If this be true of the thorax with its resilient and expansive viscera, how much more pronounced must be the permanent effect upon the pelvic framework with its non-resilient contents?

The ordinary diaper of twilled cotton flannel, firm and unyielding, folded many times, tightly drawn and pinned about the iliac crests to prevent slipping, we know too well. The tension of this ceinture is notably increased by the frequent wetting to which it is subjected. May we not establish a causative relationship between this commonest American diaper and the most frequent American pelvic deformity? Obstetricians are agreed that the dystochias in this country are largely due to justominor pelves. Is there any reasonable doubt that continuous ceinture of the pelvis during the first two years, before ossification has progressed far, will produce the justominor pelvis in women?

The great plasticity of the infant pelvis is easily seen in the moist specimen of a full term infant I now show you. When we remember that the complete pelvis is the result of development from fifty-five centres of ossification, in about a third of which the process has not even yet begun at birth, while from no one of the remaining centres has ossification proceeded to completion, that a large amount of pelvic structure, subsequently to become bone, is purely cartilaginous, the plasticity of this foundation structure of the skeleton is appreciated. Is there any doubt that continuous compression in this stage must inhibit growth? All analogies point to the contrary conclusion.

The practice, also, of wadding a large amount of inelastic material tightly between the thighs (which serves as a fulcrum when the lower extremities are firmly bound in apposition by the pinning blanket or other swaddling clothes) tends to cause deformity of the femora. This is seen in the incurvation shown by skiagrams C1 and C2. (See Figs. C1 and C2.)

There is little doubt in the mind of the writer, that many cases of genu valgum have their beginning in this cause. The align-

FIG. B1.—NORMAL INFANT THORAX.

FIG. B2.—SAME PATIENT AS B1; SHOWING THE EFFECTS OF COMPRESSION OF THE THORAX.

ment of the articulating surfaces being thus easily disturbed, the defect is naturally progressive, with the superincumbent weight of the growing body, until the age of complete ossification when the bones and joints show permanent deformity. (See Figs. C1 and C2.)

To obviate these defects the diaper should be light, with no more material than is absolutely necessary for the absorption of discharges. Absorbent cotton, either loose or in pads, preferably the latter, retained by a T bandage or triangle of some flexible material, such as cheese cloth, secured by safety pins to the shirt, before and behind, has been found to meet all the requirements.

The most prevalent method of clothing infants in continental Europe and some parts of Asia is seen in the "swaddling clothes" retained by a gasket or knitted band around the body. This binds the arms close to the body and the legs straight and in close apposition. This custom is also seen in this country in some families of foreign born people. In many instances the gasket is so closely applied as to restrain all voluntary movements except of the face.

The old-fashioned English and American costume consists of a short, sleeveless linen shirt; the "pinning blanket" or "barrow coat," a straight, firm waist band of cotton cloth, to the lower edge of which is gathered a long flannel breadth, extending eighteen to twenty-four inches below the infant's feet. The band is pinned smoothly and tightly around the thorax and the lower flannel portion folded together and brought up and secured by pins. This is followed by the regular skirt composed of flannel amply shirred to another waist band, which is also tightly pinned. Over this is a cotton or wool slip, with or without sleeves, which adds weight to the already crushed infant. Often a crocheted or knitted jacket is worn outside.

In recent years many intelligent nurses have adopted a more hygienic clothing, of which the "Gertrude suit" is a good illustration. This consists of three garments, neither close fitting shirt nor binder being used. All these garments are cut in the princess style, supported from the shoulders and extend well below the feet, the inner and outer garments having sleeves to the wrists. To this may be added short socks, and moccasins in cold weather.

To secure the benefits of clothing, and at the same time freedom from its injurious effects, is a problem, the solution of

FIG. C1.—NORMAL INFANT; PELVIS AND THIGHS.

FIG. C2.—SAME PATIENT AS A1; SHOWING DEFORMITIES PRODUCED BY A WAD OF DIAPER BETWEEN THE LEGS.

which is widely sought. The ideal protection would seem to be afforded by a large blanket of light, flexible, non-conducting material enveloping loosely the entire infant below the neck, but his normal restlessness makes it difficult to keep him within the folds of this covering. A more definite garment that cannot be thrown off, while still allowing unrestrained freedom of necessary movements, was shown by the writer before the Pediatric Section of the American Medical Association at Baltimore in 1890, and also presented by invitation to the *Société d'Hygiene Français* at Paris in 1897. This garment is in use in the infant wards of some of the hospitals of Chicago and other cities. It is a bag, so constructed that it envelops loosely the entire body below the chin, closure being secured above by safety pins and below by a draw string. Additional protection against cold is afforded by separate under garments, such as a light knitted shirt of silk or wool, free from seam or band, and one or more sleeveless slips, as occasion may require.

PILOCARPIN IN THE TREATMENT OF SCARLET FEVER.

BY E. W. SAUNDERS, M.D.,

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This drug has undergone many vicissitudes in the favor of the profession, but for a long time it has been in disrepute, owing to the deadly results justly attributed to its use, particularly in eclampsia and in the later stages of diphtheria.

In another paper* the subject of its advantages and its dangers in many diseases has been fully discussed, but here I wish to confine my attention to its use in scarlet fever, after giving a short account of its physiological effects, and the contraindications to its use in practice.

The physiological effects of pilocarpin are exerted chiefly upon the glandular structures of the secretory organs in the following order of frequency and potency: the salivary, the muciparous, the mammary, the sudoriferous, the lachrymal, the renal. Vomiting is a frequent effect, and then not only are the contents of the stomach together with saliva, which has been swallowed, ejected, but also a large quantity of mucoserous fluid secreted by the stomach itself. With a few people pilocarpin acts as an instantaneous emetic by virtue of contact with the stomach walls. The pulse, after full effects are obtained, is slower, thinner, and more compressible. The respiration is retarded and more superficial in character. Occasionally amblyopia is observed, lasting for an hour or more. In some cases, especially where a sthenic fever is present, or where an insufficient dose has been given, an increase of the pulse rate and a flushing of the whole surface may be observed as a primary effect and this may be the only result obtained. Fever exerts an inhibitory effect upon the normal physiological manifestations, and hyperpyrexia may altogether abolish them; an effect, however, not confined to pilocarpin, but observed, as all clinicians will testify, in the case of several other drugs, especially those belonging to the class of hypnotics or antipyretics. In its effects upon the kidneys it is ordinarily found

* *Therapeutic Gazette*, April, 1898.

that the solids alone are increased, but very exceptionally a polyuria is produced. Incidentally the growth of the hair is stimulated. Whether any of the internal secretions are increased is a matter of speculation, but an enhanced leukocytosis is an invariable effect, with exalted alexin formation, and most probably there is an increased activity of the toxilytic function of the liver.

It follows from the foregoing sketch, reinforced by many sad clinical experiences, that this powerful drug should not be used under the following conditions: coma or threatened coma; cardiac weakness, whether from myocardial degeneration as in typhoid, etc., or from defective innervation, as in diphtheria after the fourth day, or from lack of compensation in valvular lesions, or even when a sound heart is overburdened, as in pneumonia after the initial stage. Likewise wherever there is the least embarrassment of the respiratory function, from whatever cause, the administration of pilocarpin is fraught with danger. Lastly in the very young, in the aged, and in all cases of prolonged disease, or inanition, where the system cannot safely withstand the least depression or serous drain, the drug should not be employed.

The peculiarly beneficent effects of pilocarpin in scarlet fever are due to its effects upon the salivary and muciparous glands, coupled with the absence, generally speaking, of all tendency to myocardial or neural degeneration, and of pulmonary complications. The researches of the Pasteur Institute have well established the fact that of all the secretions, the saliva possesses the greatest toxilytic power. Therefore, whenever we can bring the saliva to play directly upon the toxins at the site of their production, we do the second best thing ideally, and the best thing practically. Not only so, but the saliva, being a normally albuminous fluid, furnishes a valuable vehicle for the excretion of toxins, which are unable to escape, as such, by any other normal route.

Whilst the saliva and the mucus are neither of them germicidal, the constant moistening of the mucous surfaces by their own secretions keeps them in the best possible condition for defense against invasion, especially of the deeper structures. While I regard pilocarpin as the most valuable single agent against scarlatinal infection, or, more strictly speaking, against the associate infections, which are probably the more pernicious, I do not by any means limit myself to its use.

In hyperpyrexia hydrotherapeutic measures are our first resource, to be followed up by the administration of pilocarpin. Even at ordinary temperatures, such as 104° F., it is better to precede each dose with a thermolytic bath. Chloral is nearly always indicated and moderate doses, frequently repeated, are best. Each dose may procure not more than one hour's sleep. The coal tar antipyretics should not be given immediately in conjunction with pilocarpin. The most recent serum treatment promises well, as does the revived nuclein administration, in vastly larger doses than those recommended by Vaughan originally; but to my mind the indications for pilocarpin will still be present, at least at the outset of every severe case of scarlet fever.

The first dose should be watched lest an idiosyncrasy be encountered. If so, then a hypodermic of atropin, "the perfect physiological antidote," will obviate all disagreeable consequences. Subsequent doses should be graduated according to the effect of the first and the height of the temperature. Toleration is rapidly established, so that the dose may have to be augmented from day to day. Even where violent vomiting is not produced, the taking and the assimilation of food may be interrupted, so that a continuous effect is not to be sought. However, a parched mouth is never to be allowed during the disease.

The beneficent effects of the drug are, moderate reduction of the temperature (when not hyperpyrexial), relief of suffering due to the parched and fissured mouth and lips, rapid improvement in the state of the oral and faucial mucous membranes, and prevention of glandular infection. If the nasal membrane be infected, there is no good result to be expected, as far as this condition and the subsequent aural complication are concerned.

A CASE OF AUTOINTOXICATION WITH NEUTRAL MAGNESIUM PHOSPHATE IN THE URINE.

BY CHARLES GODWIN JENNINGS, M.D.,

Detroit, Mich.

The following is the history of an interesting case of autointoxication: Anna J., aged ten years; a highly intellectual, sensitive child, of cultured parents. A gouty paternal history. The mother is a cultured woman of nervous temperament, who has given the most careful attention to the hygiene of her children. Notwithstanding this care, the patient during her whole childhood has been subject to attacks of acute autointoxication, rarely traceable to a definite cause. Constipation, coated tongue, nausea, vomiting, headache, dizziness and prostration have been the usual phenomena of these attacks. For the last two or three years they have been less frequent and have been made less severe or have been aborted by free elimination and abstinence from food for two or three days from the onset of premonitory symptoms. Three years ago, after one of these attacks, a persistent mild febrile movement and cough, with the physical signs of condensed lung tissue in the lower part of the upper right lobe in front, led me to advise her spending the remainder of the winter in Colorado Springs. Since that time her temperature has been quite uniformly one-half to one degree above normal in the morning and from one to one degree and a half above in the evening.

The attack reported below came after a winter of unusual freedom from digestive disturbance. It was preceded for two weeks by languor, coated tongue, indifferent appetite and indigestion.

On January 28th the bowels were freely purged with calomel. The following night she vomited. During the next morning, January 29th, she felt well, but was languid. At 2 P.M. she lapsed suddenly into a state of persistent somnolence simulating coma. She lay apparently sleeping. She could be aroused but with difficulty, and then she would scarcely open her eyes, move or speak. When fully aroused she was nauseated, dizzy and exquisitely sensitive to light, sound and movement. She vomited every few

hours. Her pulse was 120; temperature 99.2° F., respiration quiet and shallow; pupils normal.

For the next four days there was no material change in her condition.

February 3d. For about an hour in the morning, and also in the afternoon, her extremities became cool and her pulse feeble. She was very restless and in great indefinable discomfort.

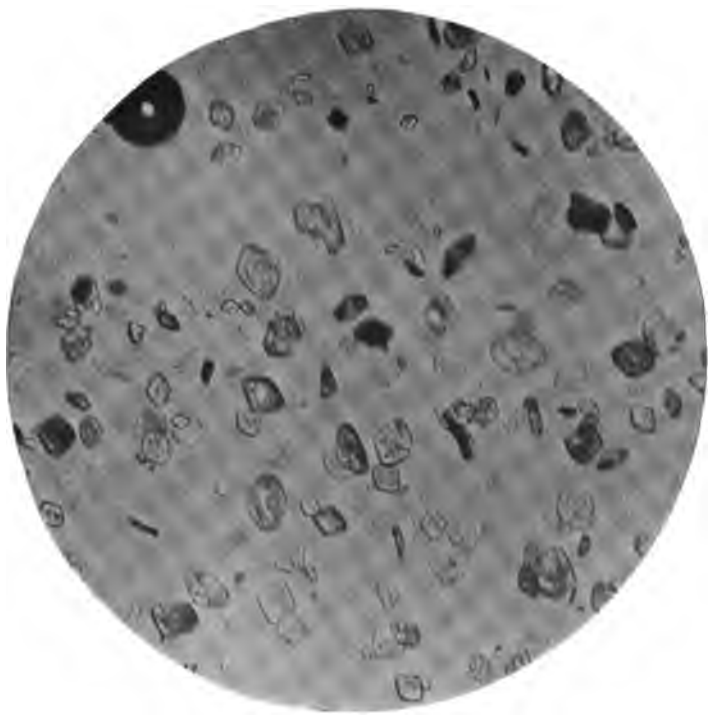


FIG. I.—PHOTOMICROGRAPH OF SEDIMENT OF NEUTRAL MAGNESIUM PHOSPHATE IN URINE.

On February 4th she had a quiet night. A free evacuation of the bowels was followed by a decided improvement in the symptoms. The patient opened her eyes, spoke a few words and moved a little. The nausea and vertigo had gone. She showed returning appetite. The pulse was 100; temperature 98.6° F.

Convalescence progressed steadily from this time, although marked by decided nervous irritability and insomnia. She was able to sit up in bed February 10th, just two weeks from the onset

of the attack. During the last week of her stay in bed her temperature remained normal.

On February 1st, a twenty-four hour specimen of the urine was analyzed. Amount in twenty-four hours, 460 cc. Color, pale yellow, cloudy. Reaction, acid. Tests for albumin, sugar, bile and indican, negative. The sediment was abundant and heavy, and appeared to the naked eye as colorless, glistening crystals. The

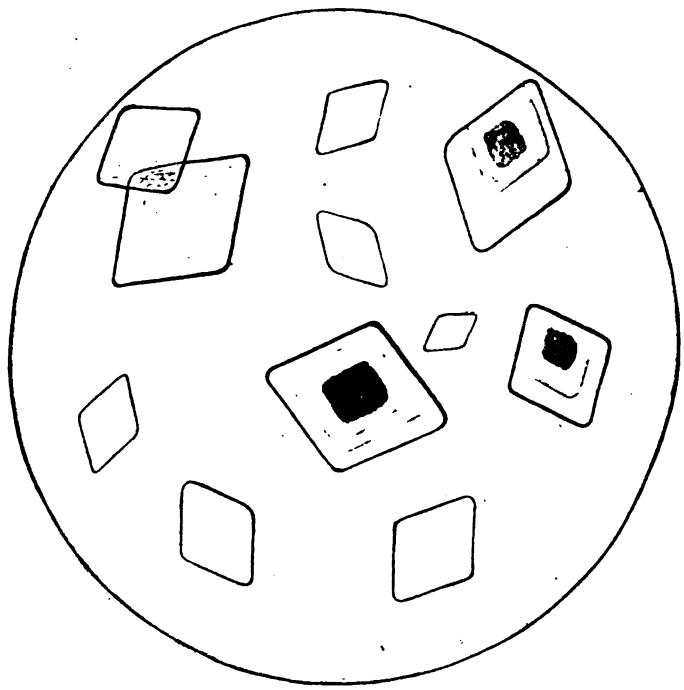


FIG. II.—DRAWINGS OF CRYSTALS OF NEUTRAL MAGNESIUM PHOSPHATE.

microscope showed the sediment to be made up entirely of highly refractive, colorless rhombic plates. Most of the crystals had irregular eroded edges, and showed on the flat surface striæ, as if they are built up of several thin plates. Neither myself nor the members of the Detroit Clinical Laboratory staff were able to identify the crystals by their morphology or by the usual microchemical tests. Chemical analysis by Dr. Stephenson, analytical chemist to the Laboratory, identified the sediment as neutral magnesium phosphate, $Mg_3 (PO_4) 2$. A photomicrograph of the

sediment is shown in Fig. I. Fig. II. is a drawing of a few isolated characteristic crystals.

This urinary sediment is of very rare occurrence, and is not described in the usual text books on urinary analysis. It was first fully described by Stein (*Deutsches Archiv. f. Klin. Med. Bd.* XVIII). According to this author it is found in concentrated,

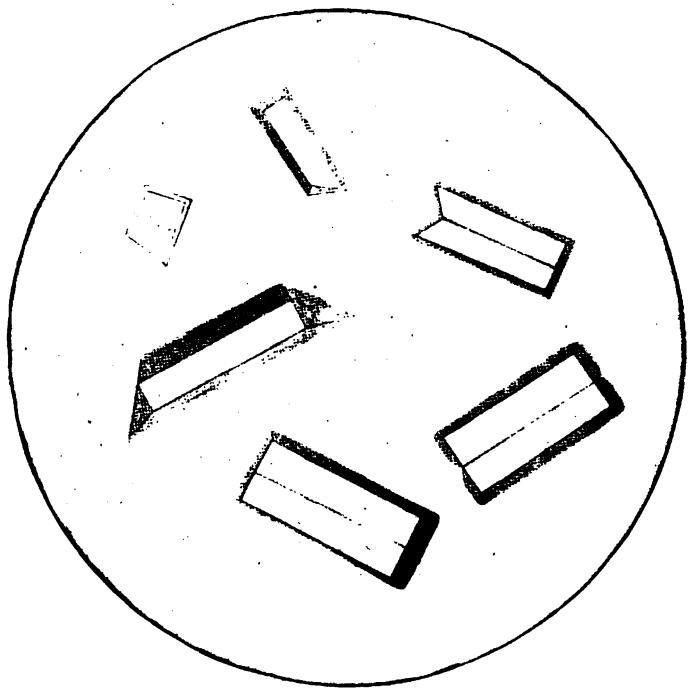


FIG. III.—CRYSTALS OF NEUTRAL MAGNESIUM PHOSPHATE, ACCORDING TO REIDER AND STEIN.

slightly acid or neutral urines in which the ammonia is not increased.

Herman Reider, in his "Atlas of Urinary Sediments," shows a plate of this deposit and states that it has been found in cases of dilatation of the stomach with pathological fermentation. He says, further, that Hassell and Golding Bird described these crystals twenty-three years before the publication of Stein's paper. The plates in Reider's atlas and in Stein's article show a different crystalization from that of my specimen when it was fresh. (Fig. III). After standing over night in the ice-box of the laboratory,

the form of crystalization changed and the morphology of the crystals was identical with that shown by Reider. The morphology of the original crystals is similar to that of certain colorless forms of uric acid (Fig. IV.), and the sediment was thought to be uric acid until it was subjected to microchemical tests.

This case is unusual and interesting both from the clinical history and the urinary findings. But one other case presenting

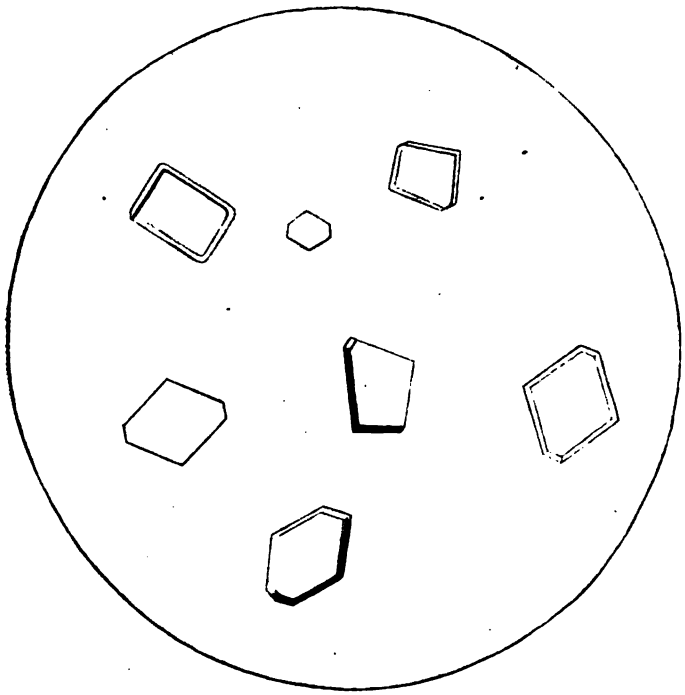


FIG. IV.—FORMS OF COLORLESS CRYSTALS OF URIC ACID.

similar clinical phenomena has come under my observation. This child, age four years, the daughter of a prominent Detroit surgeon, remained in a similar somnolent state without speaking or rousing, with quick pulse and normal temperature for five days. After what her father described as a free bilious evacuation of the bowels, the somnolence lifted, the mind cleared and she quickly convalesced.

This type of autointoxication has to be differentiated from uremia and from meningitis or other organic cerebral disease. The urinary findings would exclude uremia. The somnolence,

vomiting, constipation and vertigo are strongly suggestive of meningitis. Absence of pyrexia, vasomotor phenomena, severe headache, paralyses, slow irregular pulse and the state of the pupil and fundus of the eye, exclude organic brain mischief.

As to the significance of the magnesium phosphate in the urine, the few cases in which this sediment has been found have shown pathological intestinal fermentation and putrefaction. It should be noted, however, that in the case here reported magnesium sulphate was administered by enema several times during the course of the illness. This may possibly account for the presence of the magnesium salt in the urine.

DISCUSSION.

DR. EDSALL.—In cases of this kind, I believe it would be worth while to look for acetone and diacetic acid in the urine; because it has been established that an acid intoxication similar to that which occurs in diabetes mellitus is occasionally seen in other conditions and has repeatedly been met with in childhood. If acid intoxication were present, it would be very easy to discover it, and treatment of this acid intoxication might be of great value in obscure cases of this kind. I have recently reported a case of nondiabetic acid intoxication in an adult, in which there was a desperate, prolonged attack of coma. This man recovered entirely after the intravenous use of alkali.

DR. CHRISTOPHER.—I would like to add that personally I find high acidity of the urine often associated with autointoxication in children and that the trouble clears up under simple alkalinization of the urine, preferably with potash. I am not speaking particularly of cases of coma, but have had meningitis disappear in the same way.

DR. EDSALL.—In one case of recurrent vomiting which I saw but once, I noticed a pronounced odor of acetone at the beginning of the attack. I could not get a specimen of the urine, but ordered large doses of alkali, and the vomiting stopped at once. Since that time, alkali has at once been used in large amount when suggestive symptoms have appeared. Since that time—eighteen months ago—this child has been entirely free from attacks; while during the previous three years she had been having them very frequently.

DR. CHRISTOPHER.—I find that often in psychic vomiting they will take the bicarbonate of soda when they will not retain anything else. I had one child who took by mistake two ounces of dry bicarbonate of soda and in the morning the urine was still acid in reaction.

CLINICAL OBSERVATIONS ON CIRCULATORY FAILURE IN ACUTE INFECTIOUS DISEASE.

BY AUGUSTUS CAILLÉ, M.D.,

New York.

This is merely a preliminary report dealing with circulatory failure from (1) direct loss of blood; (2) shock; (3) loss of blood and sepsis combined (in surgical cases); (4) sepsis in acute infectious disease, based upon experiment and hospital experience.

From our knowledge of the effects of *saline infusion* in shock and hemorrhage it would appear that this procedure and also *hypodermoclysis,* may be relied upon to promptly and safely stimulate in circulatory failure. Infusion has an immediate effect; hypodermoclysis improves the circulation in from 5 to 10 minutes as shown by the quality of the pulse and the slowing of respiration. The action of both is more prompt than that of drugs and apparently increases the effect of drugs given hypodermatically. It is safe to continue with the saline until the pulse is of good quality and then to stop it.

From a study of the *septic* cases treated by saline infusion it would appear that in order to remain on safe ground it should be reserved for septic cases in which there has been a decided loss of body fluids, as in severe choleraic or typhoid diarrhea. In cases of *sepsis without loss of body fluids* and with an imperceptible pulse and rapid respiration and in conjunction with a very rapid thumping of an undilated heart, saline infusion or hypodermoclysis may be warranted, but under no circumstance should such methods be employed in a routine way or when the heart's action is hampered by pericardial effusion. Seven of the 8 septic cases, the basis of this report, were looked upon as hopeless—the ultimate recovery of 4 out of 8 was in every way satisfactory considering the gravity of the condition.

Enteroclysis properly carried out with Kemp's flexible double current catheter (15 minutes flow of water at 110° F.) appears to be an absolutely safe method of combating circulatory failure in septic conditions.

It stimulates kidney secretion and thereby promotes the elimination of poisons. It induces intestinal absorption of water whenever the body craves for water. It has a certain effect in reducing temperature. It appears to be indicated as a routine treatment in all septic conditions even if the kidneys be not involved.

In severe anemia I have found that enteroclysis is followed by an actual improvement of the blood mixture independent of the administration of drugs as shown by a number of cases under prolonged observation. In conclusion I would emphasize that circulatory failure is not always heart failure and that this distinction should receive more attention at the bedside and that enteroclysis properly carried out should be universally adopted in the practice of medicine as a therapeutic measure of undoubted value in the many cases in which increased elimination through the kidneys is desired, as in all forms of infective disease and in faulty metabolism generally. Enteroclysis may be administered alone instead of drugs or in combination with drugs.

DISCUSSION.

DR. ACKER.—I have never had a case of typhoid in a child in which I have used this treatment, but I had a moribund case in an adult where the man had had a large hemorrhage at night and was in a moribund condition the following day so that the pulse could not be felt at the wrist. Nearly a quart of saline solution was injected into the vein with immediate effect. The pulse improved, the respiration became deep and free, and there was an uninterrupted recovery.

DR. BLACKADER.—At what temperature do you use the fluid and was there any alteration in the position of the patient?

DR. CAILLÉ.—A temperature of 110 or 120 F. The buttocks are raised and Kemp's flexible tube introduced without force.

DR. BLACKADER.—In what way do you think it acts as a stimulant to the circulation?

DR. CAILLÉ.—The abdominal sympathetic is probably stimulated to some extent, and then it seems to me that if the tissues crave water it is supplied by absorption.

DR. BLACKADER.—I think that we are still uncertain as to the exact conditions under which such means will be of service, and will not merely add to the burden of an already much enfeebled heart. We know that where intravascular pressure is much lowered, cardiac action, *ipso facto,* is weakened, and it may be

that in cases where the intravascular tension is extremely low, we do increase cardiac action very distinctly by all means which increase vascular tone. This probably is the mode of action of hypodermoclysis in those cases where there has been a considerable drain of fluid from the system. In other cases the increased amount of fluid in the vessels stimulates the elimination of toxic material and lessens any depressing action arising in this way. In still others it may mean only an increased amount of fluid for the heart to drive and, therefore, I am not surprised to learn that in some cases this measure appeared distinctly injurious. The subject of circulatory failure in infectious diseases is one requiring much thought and a careful determination of the conditions present before uncertain measures are undertaken.

DR. KERLEY.—I can fully endorse what Dr. Caillé has said regarding colon flushings. I have used this treatment extensively and it seems to me that there is no danger of the fluid being taken up by the system unless it needs it. My method is to use a long rectal tube attached to a fountain syringe and introduce it until it can be felt in the descending colon when the water is allowed to flow, the bag being held about three feet higher than the child's body. The child should retain the water as long as possible. This will be best accomplished by maintaining the child in the recumbent position, with the shoulders lower than the buttocks. I have used the colon flushing with particularly good results in summer diarrhea in cases in which there had been large loss of fluid through the profuse watery discharges. A normal salt solution is always best. I have often given a pint or a quart and then measured the amount passed, and have frequently found that as much as ½ pint has been retained. During the past winter I used it in three acute septic cases in which it proved signally beneficial, from three to four of the colon flushings were given daily, about one-half of the fluid being retained and absorbed into the circulation. I have not used the solution as hot as Dr. Caillé recommends excepting in the cases in which there were great prostration and low temperature. In the diarrheal cases in which there was high fever I have used it as low as 70 degrees, with the idea of aiding in the reduction of the temperature. In the septic cases it was usually used at the body temperature. It has also been of service in the acute infectious diseases in which there was diminished renal secretion. In acute nephritis or in post scarlatinal nephritis it is of much service in starting up the kidney function. To my mind it does better execution as a diuretic than any other single measure we have at our command.

DR. SEIBERT.—I simply want to say that as some of you may know, during the last fourteen years I have made use of systematic enteroclysis in the treatment of typhoid fever patients. In

severe cases with very high fever I give an enema every six hours, in adults, using a quart and a half, but never with the catheter, because of the danger of coming in contact with typhoid ulcers in the rectum. In mild cases enemata are given twice daily, simply elevating the buttocks. If some of the water is retained it is all the better for the patient. I have published on this subject as long as twelve years ago. As regards its use in the gastro-enteritic cases of children I endorse what Dr. Caillé has said.

DR. JACOBI.—One factor in these cases is the filling of the blood vessels with a circulating fluid when it is wanted. When that is so, the saline solution will do at almost any temperature, that of the blood or a little less. Dr. Caillé has told us that he has used injections of 110-120° F. At that temperature it acts as a stimulant. He has spoken of the stimulation of the splanchnic nerve; shock means paralysis of the splanchnic and that means accumulation of blood in the vessels of the abdominal cavity to such an extent as to leave the rest of the body anemic. By stimulating the splanchnic nerve with this heat you supply the body with blood again and you have the fluid you inject and this blood that has been stored away added to the circulating medium, and, I think, that explains in part the rapid effect produced.

DR. COTTON.—When listening to Dr. DaCosta I remember he made an emphatic point, clinically, of enteroclysis in colitis. Asafetida, turpentine and guiacum were added to the enema. I followed his advice eighteen years ago and met with such success in cases of exhaustion from this cause that perhaps I gave too much credit to the drugs used, until within the past six or seven years the normal salt solution has taken the place of the drugs. Still I must admit that I believe that these drugs will help to accomplish what Dr. Jacobi has attributed to the heat.

DR. ACKER.—There are two men in Washington who have done much work in this direction, Drs. Bovée and Stone. Dr. Bovée has devised a thermometer for recording the temperature of the water as it enters the bowel and he found that there is always a loss of 10 or 15°. Dr. Stone has done some interesting work, too, along this line, using intravenous injections.

DR. ADAMS.—The loss of heat would depend upon the length of tube used and the rapidity with which it passed through. It certainly should not lose 10 degrees.

DR. ACKER.—If it is desired to use water of 110° Dr. Bovée begins with a temperature of 120° in the bag, allowing for a loss of 10°.

SUDDEN DEATH OF AN INFANT SIX MONTHS OLD DUE TO COMPRESSION OF LARGE BLOOD VESSELS BY AN ENLARGED THYMUS GLAND.

BY AUGUSTUS CAILLÉ, M.D.,
New York.

Intra vitam the infant was occasionally slightly cyanotic and had spells of rapid and laborious breathing. On two occasions it had convulsive seizures. Auscultation revealed a loud systolic murmur at the base of heart with the valve sounds clear and distinct. The murmur was not transmitted in any direction. The infant died suddenly and the autopsy showed, as was suspected, a large thymus gland measuring two inches in length and one inch across. The lower pole of the gland compressed the large vessels in such a way as to cut off the circulation.

DISCUSSION.

DR. JACOBI.—Here was a child that showed no other symptoms, no heart disease, and who died suddenly with symptoms of cyanosis. Nothing else was found but a large thymus gland, an unusually large thymus. There are undoubted cases in which babies die this way. I had a paper on this subject in the third volume of the Transactions of the Association of American Physicians. The slightest increase in such a thymus gland would certainly cut off respiration. An impediment to respiration caused by such a large gland would be perhaps endured for a time and then it gets so large that it takes but the slightest increase to kill the baby at once. The distance between the manubrium sterni and the vertebral column is little more than 2 cm.: the space which is filled by esophagus, trachea, blood vessels, nerves, connective tissue, etc., becomes very narrow and congestion of a few seconds will so further narrow it that suffocation is possible. That is what I believe takes place in these cases, not to speak of the possible influence on the recurrent and other nerves.

DR. CAILLÉ.—It was clear to us that the thymus gland pressed upon the large vessels from the heart and that there was complete stoppage.

DR. JACOBI.—These cases need not be called acute, but when we consider the narrowness of the space in which so many tissues are found it is quite probable that a very trifling addition to the amount of blood in the thymus itself will so narrow the space that circulation is impeded. Only a very few cases have been reported since Kopp reported his first case, nearly a hundred years ago, of thymic asthma.

DR. PUTNAM.—A case of death occurred under such circumstances at our asylum in Boston a few months ago. The child was in its crib and no one looking at it at the time. The nurses supposed that it had turned on its face and suffocated. We had an autopsy and nothing was found but a large thymus gland which weighed six ounces.

DR. JACOBI.—There is a case on record by Koenig in which symptoms of suffocation existed, the diagnosis of enlarged thymus was made and part of the thymus removed, with life saving effect. The thymus was drawn out and fastened in front, away from the blood vessels and larynx, by Rehn and by Koenig, and the infant got well. That [illegible] ne to be absolute proof of the cause. [illegible] n the neighborhood gave relief. [illegible] edition of Therapeutics. The [illegible] st cases. It is quite possible by [illegible] positions. When the body is on [illegible] can be percussed. Sometimes, [illegible] gives marked dullness and in an [illegible] crease considerably. If you per- [illegible] g supported face downward, the [illegible] large surface, and, one like this [illegible] percussed in that position. I [illegible] ave seldom failed to make a [illegible] n published in the last year [illegible] speaks of the possibility of [illegible] nus. In my first case of the [illegible] just died. Nothing was to [illegible] n, and I percussed the chest [illegible] that there was some dullness and I suggested the possibility of enlarged thymus. I turned the baby over and then I got a large extent of dullness and fullness in that neighborhood. Since then I have frequently done the same thing.

REPORT OF A CASE OF SINUS THROMBOSIS RESULTING IN EXTENSIVE CEREBRAL HEMORRHAGE IN AN INFANT FIFTEEN DAYS OLD.— SIGMOID FUSION OF THE KIDNEYS.

BY SAMUEL McC. HAMILL, M.D.,
Philadelphia.

The infant from whom these specimens were removed was born on the 10th of January, 1902; there was no history or evidence of syphilis in the mother; the labor was normal and the child was not asphyxiated. His temperature was slightly elevated from the second day; on the seventh day he developed a papular eruption on his face. The infant's temperature rose suddenly with the appearance of the eruption to 104°F. He took nourishment well and had no gastrointestinal disturbance. The temperature fluctuated between 101° and 104° F. during the eighth, ninth, tenth, eleventh and twelfth days of life. The facial eruption persisted, and a similar eruption developed over the neck, shoulders and flexor surface of the arms. The persistent fever was attributed to this eruption.

On the thirteenth day "the baby gave some slight evidences of pneumonia," rapid respiration and a harsh vesicular murmur. The temperature became a little more elevated. A cotton jacket was applied.

On the fourteenth day there developed marked twitching of the muscles of the right eye and constant lateral rotation of the head. There were no general convulsions. The child did not cry out. He had no gastrointestinal symptoms. He nursed poorly and was somewhat cyanosed.

On the fifteenth day the temperature was less high—102°F.—and he seemed much better. The movements of the head had almost ceased, and the twitching of the right eye was less; nourishment was well taken. The eruption had become more marked and in some places pustular.

On the sixteenth day, the day of death, the temperature rose to 106.8°F. at 8 A.M. The child vomited a large quantity of

blackish material, which was evidently altered blood, and had several tarry stools. The face became much cyanosed; great restlessness ensued, and death occurred rather suddenly at midday.

AUTOPSY.

The autopsy was made about twenty hours after death. The body was poorly nourished; the head was large, the fontanel bulging. The cranial bones were widely separated in the lines of the sutures.

A papulopustular eruption was present on the face, neck, forehead, shoulders, arms and upper part of the chest; in some areas it was hemorrhagic, especially over the flexor surfaces of the arms. Postmortem lividity was present in the dependent portions of the body. Rigor mortis was marked. The umbilical stump was not entirely healed and rather unhealthy in appearance.

Abdomen.—On opening the abdomen the umbilical arteries were found to contain thrombi. The stomach was much distended and the liver enlarged, extending below the transverse umbilical line. The lobus Spigelii seemed abnormally large. The gall bladder was partially filled. The spleen was slightly congested, but not enlarged. The kidneys were anomalous. The cecum and small intestine overlaid the lower portion of the anomalous kidney, and the vermiform appendix was closely applied to its anterior surface. The ascending and transverse colons passed over and were adherent to its upper portion. There was some congestion of the vessels of the mesentery, and some enlargement of its lymph nodes.

Thorax.—Examination of the thorax showed a half ounce of clear liquid in the left pleural cavity. The thymus gland was not enlarged. The pericardium contained a half ounce of clear fluid; its walls were smooth; both auricles were distended with liquid blood, the right more than the left. The veins of the heart were greatly distended. There were no abnormalities in the heart.

The lungs were free and showed some congestion at their margins.

Abdominal Organs.—The stomach contained some blackish material having the appearance of altered blood. There was no congestion nor erosion of its mucous membrane. The intestines contained nothing abnormal. The liver on section was yellowish in color and rather soft. The spleen was normal.

Kidneys.—There was no kidney present on the left side. The left suprarenal gland occupied its normal position and was normal in size and appearance. There was a fusion of the kidneys of the sigmoid variety—the anomalous kidney lying entirely to the right of and close to the vertebral column. There was no other abnormality found.

The Brain.—On opening the cranium, following the lines of the sutures there was found, overlying the left hemisphere, a large mass of clotted blood, more marked anteriorly. This extended into the substance of the brain causing some destruction of tissue. Overlying the right hemisphere there was a less marked hemorrhage. The superior longitudinal, the left lateral and the straight sinus and the vena magna Galeni contained firmly organized thrombi. The veins of the brain throughout were distended with blood, and some of the superior cerebral veins on the left side contained thrombi; both lateral ventricles were dilated and contained considerable blood, especially the left.

Cultures were made on blood serum from the thrombus in the umbilical artery and from the spleen. There was a moderate growth on each tube. Cultures from the heart blood were contaminated. Agar slant transfers showed profuse growth after twenty-four hours; one showed a pure growth and the other an almost pure growth of a bacillus, possessing practically the same cultural characteristics as the colon bacillus.

The extensive thrombosis and hemorrhage found within the cranium is of considerable interest. The history of the case, the temperature record, the result of the bacteriological study, demonstrating a short, rather thick bacillus, closely simulating the colon bacillus, and the unhealthy appearance of the umbilical stump mark this as one of those not uncommon cases of infection of the new-born.

It has been shown pretty definitely that thrombosis never occurs without some previous disturbance of the vessel wall. In the veins it is a phlebitis which produces the thrombosis and this phlebitis is usually of microbic origin. This was the probable cause of the extensive thrombosis of the dural sinuses in this case, since, as has been said, the whole aspect of the case suggests an infectious condition. The extensive subdural and cerebral hemorrhages were probably secondary to the sinus thrombosis, that of the superior longitudinal sinus and cerebral veins accounting for

the subdural and superficial cerebral hemorrhages, and that of the straight sinus and veins of Galen for the hemorrhage into the ventricles.

While it is possible that all the instances of socalled hemorrhagic disease of the new-born are not infectious in origin, the vast majority are so unquestionably. The source of the infection has never been satisfactorily determined. The condition is almost entirely an institutional disease. One rarely runs across cases in private practice; on the other hand, most institutions have had experience with the condition, and usually in an epidemic form. It does not seem to bear any close relationship to septicemia in the mother. The latter condition exists frequently without the former, and epidemics of the infantile disease develop where there have been no cases of maternal septicemia in many months.

Renovation of the hospital wards will oftentimes entirely eliminate the disease. In a very serious epidemic in the Maternity Department of the University of Pennsylvania a few years ago, in which about 20 per cent. of the babies died, it was discovered that the infants became infected in a room, devoted exclusively to the use of the babies. Attention was directed to this room by the fact that children sent to their homes in a wretched condition improved immediately. The closing and complete renovation of it instantly checked the development of new cases.

Bacteriological studies have shown that there are a number of different microorganisms capable of producing these infections. In the University Maternity epidemic, bacteriological studies were made by Riesman (University Hospital Pathological Reports) and in nearly every case cultured the staphylococcus albus or aureus, one or both were found.

Nicholson (*American Journal Medical Science,* October, 1900) reported a case infected by the bacillus pyocyaneus. A number of instances are recorded of infection by the colon bacillus, the staphylococcus albus and aureus; some with the streptococcus, the bacillus lactis aerogenes, the bacillus of Friedlander, and some with a bacillus described by Gaertner as closely resembling the colon bacillus, and probably one of its many varieties. I have found in several instances bacilli which resembled the colon bacillus in nearly all their characteristics, and yet were dissimilar in a few particulars.

From one hemorrhagic case I recovered a bacillus which was pathogenic to guinea pigs, and which produced congestion and hemorrhage into some of the abdominal organs. This resembled the colon bacillus in every respect save that it was nonmotile. I, therefore, classified it as the bacillus coli immobilis. In the case which I here report the bacillus closely resembled the colon bacillus, but was not exactly like it in microscopic appearance, and it did not produce indol.

The port of entry of the organism is as yet undetermined. It is probable that, in many instances, it is through the umbilical stump. In the case here reported, for instance, the unhealthy stump suggests such an origin. The gastrointestinal tract seems to be the avenue sometimes. In a series of cases which I observed in one of the lying-in hospitals of Philadelphia, the first symptoms noted were intestinal. It is not improbable that the infecting organism is sometimes introduced during the passage of the head through the birth canal. The skin and respiratory apparatus are sometimes held accountable, but I am inclined to think instances of infection through these media very infrequent.

NOTE ON THE TEMPERATURE CURVE OF ACUTE CROUPOUS PNEUMONIA IN CHILDREN.

BY CHARLES GODWIN JENNINGS, M.D.,
Detroit, Mich.

I present to the Society charts of the temperature, pulse and respiration of five successive cases of acute croupous pneumonia that have come under my observation during the past year. The cases were all in private practice, and were under the care of educated nurses from the inception of the disease. In no instance was the temperature influenced by antipyretics. The charts illustrate a

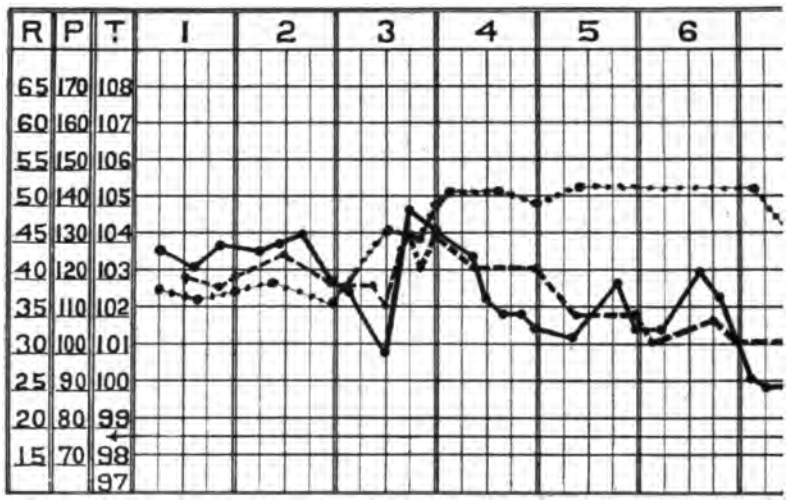


CHART I.

sharp remission in temperature that I have frequently noted in the first two or three days of the course of the disease in children, and which, in cases with tardy development of physical signs, has been a very confusing element in diagnosis. A number of times I have been misled by this unexpected drop, and have had my attention directed for a few hours away from the lungs. The recognized typical temperature curve of croupous pneumonia shows a sharp rise at the onset of the disease, and but slight variations in the curve until the crisis or pseudo-crisis.

In these charts, the heavy line is the temperature curve, the line with dashes is the pulse curve, and the dotted line the respiration curve.

CHART I.—A typical case of croupous pneumonia in a boy aged

four years. Infection of the right upper and middle lobes. Crisis at the end of the sixth day. At the end of the second day the temperature dropped from 104°F. to 100.6°F. With this drop of temperature there was no marked change in the respiration or pulse curve. At the end of the remission, however, both pulse and respiration sharply rose. The diagnosis in this case was plain from the first day.

CHART II.—Croupous pneumonia of the left apex, in a girl of five years. Crisis on the sixth day. Physical signs of incipient

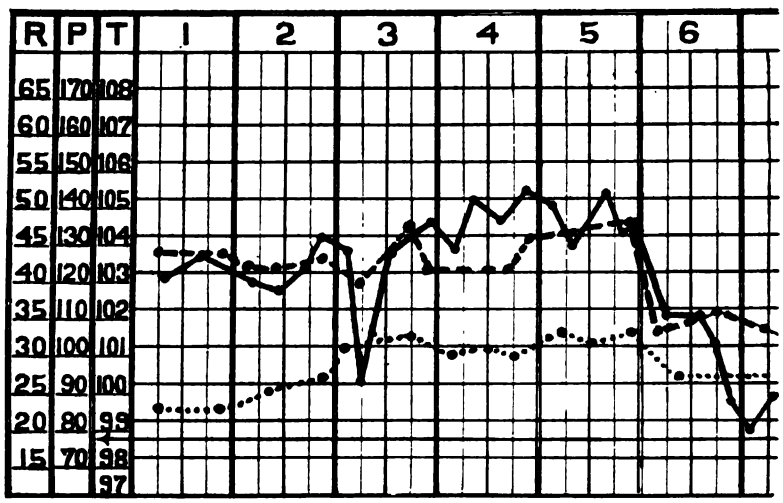


CHART II.

pneumonia present at the end of the first day. At the end of the second day or beginning of the third there was a drop in the temperature from 103.6° F. to 100° F. No corresponding drop in the respiration and pulse curves. As will be seen from the chart, the respiration in this case did not show the usual rapidity. For the first two days it did not rise above 25, and the maximum during the course of the disease was 32.

CHART III.—Acute croupous pneumonia in a girl three years of age. The appearance of the physical signs in this case was delayed until the fifth day (marked on the chart "D") and a diagnosis was not made until this time. The patient was under the care of another physician, who was completely misled by the remission that came on the third day. The temperature then

dropped from 105.2°F. to 99°F. This case terminated in lysis, and convalescence was protracted.

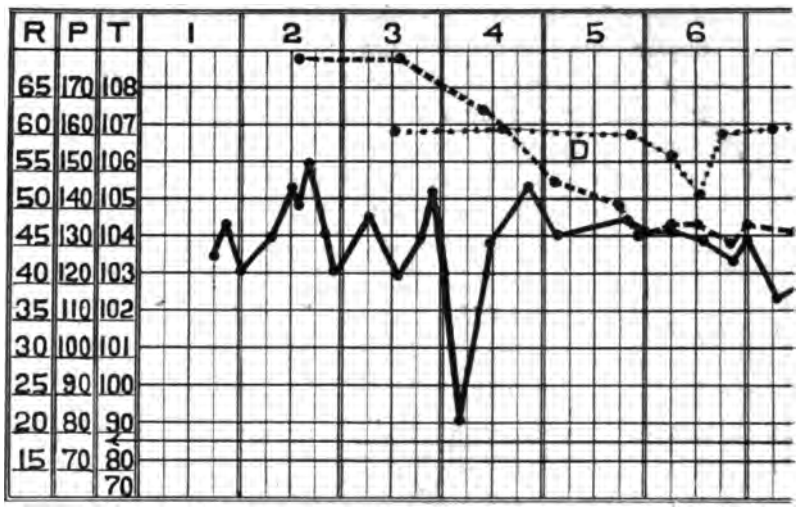


CHART III.

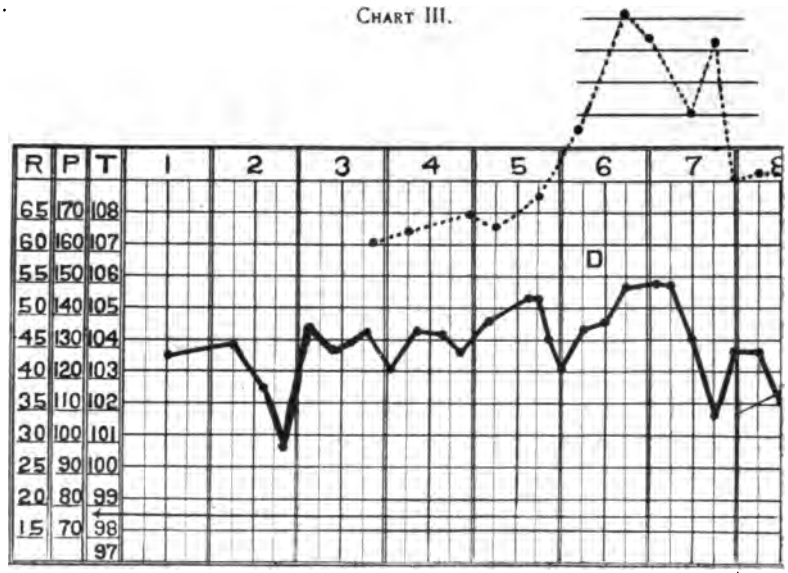


CHART IV.

CHART IV.—An infant aged one year. Infection of right apex. Crisis on the eighth day. At the end of the second day

the temperature dropped from 104° to 100.6° F. The case occurred in July, and was diagnosticated as acute gastroenteric infection. The drop in the temperature on the second day was the cause of the confusion in diagnosis, and the diagnosis was made by the physical signs on the sixth day.

CHART V.—A boy, aged five years. Infection of right lower lobe. A sharp remission at the end of thirty-six hours, the temperature dropping from 104.5° to 101.2° F. The drop in the temperature and the general amelioration in the symptoms that came with it threw me completely off the track of pneumonia,

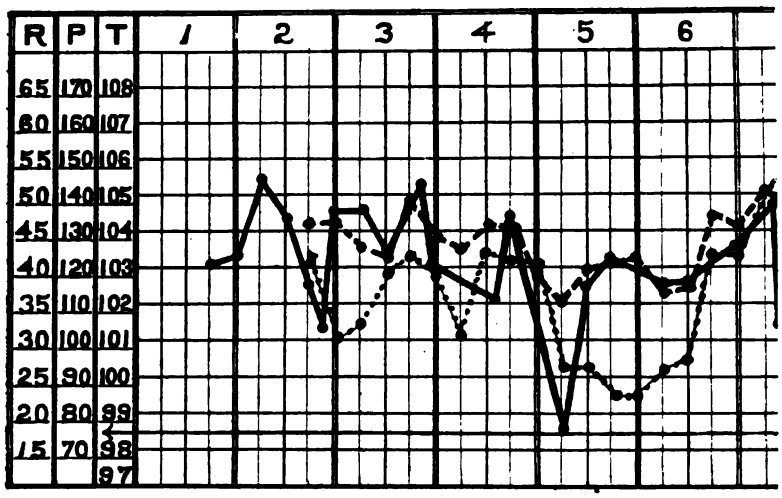


CHART V.

and the diagnosis was not made until the following day. The physical signs appeared on the fourth day. A pseudo-crisis came on the fifth day with a corresponding drop in respiration. The case terminated in empyema, with recovery.

In 3 of these cases, then, the sharp, early remission in the temperature confused the diagnosis, that in 2 cases was corrected only by the late appearing physical signs.

DISCUSSION.

DR. MORRELL.—As I look at these charts I can recall that it is not an uncommon occurrence to see this temperature drop during the first three or four days of a pneumonia. It is a fact that may be of use to all of us where the diagnosis is obscure and I thank Dr. Jennings for calling our attention to it.

A CASE OF PNEUMONIA, PROBABLY DUE TO STREPTOCOCCUS INFECTION.

BY WALTER LESTER CARR, M.D.,

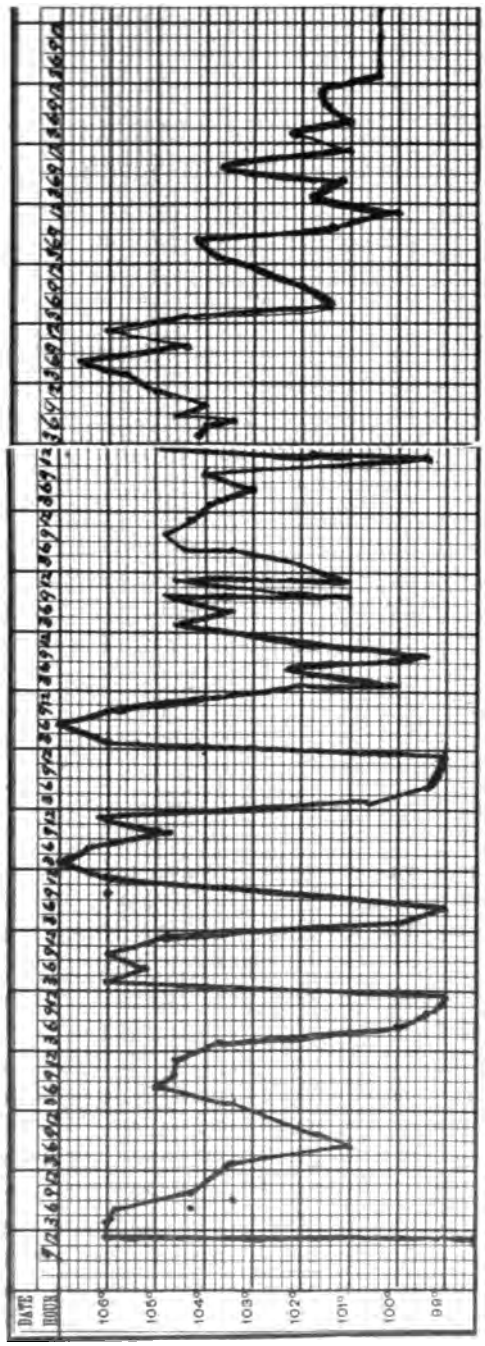
New York.

The history of this case of pneumonia is unusual in the family record and the temperature range.

M. F., a female infant of eight months was seen in consultation with Dr. Leonard S. Rau, on April 15, 1902. The notes furnished me by Dr. Rau are as follows:

FAMILY HISTORY.—Parents American, in good health. Two brothers of the patient *aet.* eight years and four years and six months are well and strong. The paternal grandfather died of general sepsis, the result of a cut on the tongue received in sealing an envelope. A paternal aunt had an attack of acute osteomyelitis of the femur develop after a slight operation on the nose. She ultimately recovered after a severe septic state with meningitis and pericarditis. The history of her case was reported by Dr. Howard Lilienthal in the *Medical News,* July 17, 1897. An uncle had an attack of erysipelas which ended in recovery.

PREVIOUS HISTORY.—The baby was bottle fed with fresh cow's milk diluted, and was in excellent health until six weeks before her present illness, when she weighed 17¼ pounds. At that time she had a foul smelling discharge from the left ear which was relieved by irrigations of creolin solution. One week before the illness here recorded the baby was restless and feverish. The discharge from the ear returned and she was seen by Dr. A. N. Strouse who found that there was a purulent discharge coming from the auditory canal and also some discharge from the nose. The tympanum showed a small perforation. The nasopharynx contained a few soft vegetations which were easily broken with the finger. Three days later the baby was again seen and the ear found to be doing well, but the other tympanum was congested. The drum was perforated and a drop of bloody serum exuded. The nasopharynx was clear and the nares were not discharging.



TEMPERATURE CHART. CASE OF SEPTIC PNEUMONIA.

On the following day both ears seemed well. Although the patient had had a restless and feverish night nothing in the examination of the ears accounted for it and the temperature was 98 $^{5}/_{10}$° F.

In the interval between the two attacks of inflammation of the ears the baby had a laryngeal seizure of a croupy type which lasted about forty-eight hours. She continued to take food and did not show symptoms referable to other organs.

PRESENT ILLNESS.—April 12, 1902. The rectal temperature in the morning was 105°F. The baby was restless all night and did not sleep. After a rectal irrigation the temperature was 99.2°F. No result from irrigation.

April 13th. The baby had a bad night. Morning temperature in rectum 104.2°F. Afternoon temperature 98.6°F. Irrigation of the intestine did not bring away undigested material or mucus.

April 14th. Baby had a more comfortable night. Morning temperature 102.4°F. Negative results in the examination of organs.

April 15th. The night was a restless one without sleep. Morning temperature 106°F. in the rectum. Examination of lungs negative except for a small area of dullness over the base of right lung in posterior axillary line. A few fine crepitant râles present. No cough.

When I saw the baby she was pale and cyanotic with a pulse of 148 and respiration of 32. This disproportion in the ratio was observed through a great part of the illness. The liver and spleen were normal and the baby's nutrition and general vitality were remarkably good. No cerebral symptoms were observed.

The patient was carefully nursed and was seen in consultation by Drs. A. Jacobi and I. Adler. The history of the case is best shown in the chart (See Chart) but a few explanatory notes may be added.

On April 18th, Dr. F. E. Sondern got a culture from a throat swab and found colonies of streptococci and of Friedlander's pneumococci. In view of the rapid rise and fall of temperature and because he detected a slight enlargement of the spleen, Dr. Jacobi examined the blood for plasmodium malariæ but the results were negative. Unfortunately, blood cultures were not taken as the baby's collapsed condition when Dr. Sondern made his visit and family reasons prevented.

The Urine by catheter contained albumin and granular casts, with epithelial detritus from the kidneys.

From the detection of the pneumonic process on April 15th to the time of the baby's death on April 25th, the disease progressed steadily. The small area of inflammation in the right lung was followed by similar patches in the left base. At first these areas were confined to the lower part of the lungs but later the upper lobes became involved and the dullness showed that the patches coalesced. The percussion note over the anterior part of the chest was resonant for some days after the dullness in the axillary and suprascapular regions was noted. The cough was not especially irritating.

The Respirations were as low as 30 and as high as 80. The latter was counted only once or twice. The respirations were 44 with a temperature of 106° and 32 with a temperature of 98.6°, while with a temperature of 107.2° on April 18th they were 48. Late in the disease the respirations were often labored and showed a Cheyne-Stokes' character.

The Temperature had a regular rise after the evening of April 15th, with the maximum temperature in the early morning and the minimum in the afternoon. The highest temperature recorded was at 3 A.M. on April 18th, when it reached 107.2°F. in the rectum. A normal temperature was recorded on April 17th, 18th and 19th, about midnight, when it would begin to rise to the morning acme. During the periods of high temperature the baby was blue and very uneasy.

After April 20th, the temperature was more irregular and the pneumonic signs were easily detected. Each rise of temperature seemed co-incident with an extended involvement of the lungs.

The Pulse Rate varied between 116 and 180. It was usually of a good quality, but at times it was fluttering and irregular in force and frequency. The tension was low. The highest pulse rate was after midnight when the weakness and general cyanotic state were such as to require the most energetic stimulation. The patient would perspire freely when asleep.

The Nervous System was not especially excited during the course of the disease. Early in her illness the baby was quiet and playful except when in pain from her ears. Later in the illness she was restless, moaned and tossed herself from side to side. The

reflexes were slightly exaggerated and there were twitchings of the hands.

Slight strabismus of a transient character was noted. The pupils were usually normal but were also observed to be dilated.

The Digestive Organs gave little trouble for the first days of the illness. The baby enjoyed her milk and took it well. She had slight abdominal distention. Later in the course of the disease the movements showed mucus and casein and there was considerable flatus. The abdomen became tympanitic. Coughing would cause vomiting of undigested masses of milk. The food was predigested and diluted according to the requirements.

The spleen was palpable for two days. It could be felt about one finger's breadth below the free border of the ribs. It did not become enlarged at the end of the illness. The liver was normal.

THE TREATMENT in this case did not give positive results. The greatest comfort to the baby was from packs at a temperature of 70°F. and baths at 85°F. Alcohol sponging was also used.

Unguentum Credé, antistreptococcus serum and hypodermatic injections of quinin dihydrochlorid carbamate were unavailing. and no effect was produced by them. The antistreptococcus serum caused a great deal of discomfort and shock, which latter may have been a coincidence and due to the disease. Glonoin and strychnia were administered as indicated and oxygen was given as required.

No autopsy was allowed.

DISCUSSION.

DR. ADAMS.—How do you account for the periodical rises of temperature? It looks as though quinin might have done some good.

DR. CARR.—Quinin was given hypodermatically, but without effect. The blood was examined with negative results. The spleen was palpable for about two days. The pneumonic inflammation was the cause of the periodical temperature.

DIPHTHERIA WITH PERSISTENT TRISMUS AND OPISTHOTONOS.—ESCHERICH'S PSEUDOTETANUS.

BY IRVING M. SNOW,
Clinical Professor Diseases of Children, University of Buffalo, Buffalo, N. Y.

In 1897[1] Escherich described a curious symptom-complex, consisting of persistent generalized tonic contractures of the muscles of the jaw, neck, back, and legs. To this condition he gave the name of pseudotetanus, classifying it as a rare type of tetany. This pseudotetanus in its clinical history bears much resemblance to true tetanus, having in common the symptoms of trismus, opisthotonos, muscular crises, and laryngospasm. It may occur as an independent malady or associate itself with an acute infectious process, as diphtheria, scarlatina, grippe or otitis media. Nearly all of the recorded cases have at first been diagnosed as tetanus. Nevertheless, although the onset of the disease is acute, it quickly assumes a more tranquil type; and although the same group of muscles may be affected in false and true tetanus, the course and termination of the diseases are widely different. It is quite probable that many cases of socalled idiopathic tetanus ending in recovery are merely examples of this interesting phase of tetany.

No writer upon tetany has a wider experience or greater knowledge than Escherich, of Gratz. His material, amounting to hundreds of cases, has been carefully analyzed, and a masterly treatise upon tetany was read by him in Moscow in 1897. He states that tetany is a condition with a free sensorium, no fever, and consists of tonic contracture of groups of muscles; that it is independent of rachitis; that laryngismus is a tetany of the laryngeal muscles often associated with other signs of tetany.

Two forms of the disease are seen: (1) Intermittent muscular contractures; (2) persistent muscular contractures.

The intermittent form, observed usually in rachitic children, is characterized by short, painful cramps, heightened mechanical and electrical excitability, and acute or subacute course, with a tendency to recurrence. Trousseau's and Chvostek's signs are easily elicited. On the contrary, in what Escherich calls the per-

sistent or tranquil type the contractures affect the trunk or extremities, run a painless and chronic course, continuing even in sleep for weeks or months. The especial nerve excitability—Trousseau's, Chvostek's, Erb's symptoms—may be lacking. The persistent contractures may involve single muscles, be localized in the extremities, may affect the entire body, may occur by themselves, or as a complication of various affections. When the tonic muscular spasm involves principally the masseter and dorsal muscles Escherich gives to that condition the name of pseudotetanus.

The onset of pseudotetanus is usually abrupt. A child either previously well or suffering from some acute infection or toxemia is attacked with stiffness in the legs and rigidity of the jaws; the contracture rapidly extends until the patient lies helpless in bed with trismus, opisthotonos, and rigidity of the extremities. The arms may be spared or very lightly affected; the muscular crises (acute muscular spasms) are usually mild and few in number as compared with tetanus. The classical signs of tetany—Trousseau and the facial symptoms—may not be present. The malady lasts from one to six weeks, the patient comfortable, even cheerful, in his unnatural posture. All of the cases of pseudotetanus reported, in spite of threatening laryngismus and intercurrent disease, made complete recoveries.

In Allbutt's[2] *System of Medicine,* Rissen Russell, writing of tetany, mentions gradations in muscular contracture from carpopedal spasms to those simulating tetanus. He states that the poison of tetany acts upon the neurons of the medulla and anterior horns of the spinal cord, damaging their functions and nutrition; that all neurons are not equally affected; that in different cases varying groups of muscles may be involved, and that in some cases a wide implication of nerve cells may exist, extending even to the motor neurons of the cerebral hemispheres. Russell says the masseter, cervical, and dorsal muscles may be attacked, producing trismus, and opisthotonos suggestive of tetanus, but differing from that disease in the happy issue of the malady.

Escherich has described a pseudotetanus of the newly born, with opisthotonos and recurring arrest of breathing. Of this type I reported 2 cases in the ARCHIVES OF PEDIATRICS, October, 1901, under the title of "Acute Recurring Respiratory Failure in the Newly Born."

My interest in pseudotetany was awakened by attending a mild case of diphtheria associated for many days with the very singular complications of trismus, opisthotonos, spasms of the muscles of the extremities, and laryngismus. This condition, which I at first called tetanus, I shall now classify as Escherich pseudotetanus.

St. Ange Roger[3] in his *Thesé de Paris,* 1902, on "Rare Forms of Infantile Tetany," has collected 9 cases of pseudotetanus in children. Of the 10 cases—St. Ange Roger's and my own—nine were boys, one a girl. The ages varied from four and a half to nine and a half years. All of the children had the symptoms of trismus opisthotonos—tonic spasms of the legs—and all recovered.

In 4 cases the disease existed by itself, 2 were complicated by diphtheria, 1 by influenza, 1 by scarlatina, 1 by intestinal worms, 1 by otitis media and rhinitis. In 2 cases the facialis and Trousseau symptoms were present, in 1 absent, and 1 not mentioned.

My patient was a Polish boy, aged seven years, of strong, vigorous physique. About August 25, 1901, it was stated that he ran a sliver of wood into the sole of his right foot. This accident was afterward denied, and no injury was found in his foot at any time. August 31st the boy complained of sore-throat, and was seen by the family physician. The doctor discovered a diphtheritic tonsillitis and pronounced rigidity of the spine; a day later the jaws grew stiff, and the child was unable to open his mouth. The muscular contracture which was first observed in the back, then in the masseters, lastly invaded the extremities, the legs became extremely rigid, and the arms slightly so. On the sixth day of the disease there appeared frequent attacks of laryngismus. The boy would cease breathing, grow black in the face; the arms and legs would stiffen out, and the trunk would arch backward, so that the patient touched the bed only with his occiput and heels. These muscular crises were frequent and severe, so that on the sixth and seventh days of illness the physician in attendance counted four in one-half hour. The muscular contractures were painful, and were often caused by handling or efforts at treatment. Thus after a week's illness, in addition to the tonsillar diphtheria, there had developed violent generalized tonic muscular spasm of the jaws, neck, trunk, extremities, and respiratory tract. No fever. Tetanus was diagnosed, and the boy entered the Buffalo General Hospital September 7th.

The resident physician, Dr. Leonard, gave chloroform to relax the jaws, and examined the throat. He perceived a diphtheritic exudate on the tongue, tonsils, cheeks, and roof of the mouth. The tongue was enormously swollen and bulging between the teeth; its tip was literally hanging in shreds, evidently lacerated by the teeth in a spasmodic closure of the jaws. There was a purulent nasal discharge and swollen submaxillary nodes; cultures from the nasal discharge, tongue, and throat showed the diphtheria bacillus. Diphtheritic antitoxin was administered; a slight amount of fever present disappeared, and the pulse and temperature remained normal throughout the disease. After the chloroform narcosis had passed away the trismus opisthotonos, and rigidity of the extremities returned. Violent muscular tetanic paroxysms, with opisthotonos and cyanosis, occurred in the morning and afternoon of September 9th; 5:30 A.M. September 10th, and 3:30 A.M. September 11th, when these symptoms finally disappeared, and he passed into the tranquil type mentioned by Escherich.

I first saw the patient September 11th. He was well nourished; clear mentally; fairly comfortable in mind and body. He lay upon the abdomen, with the head slightly retracted, but easily movable; no ocular symptoms, jaws tightly closed, masseters hard and firmly contracted; the face wore the grin of lockjaw. Several teeth gone; tongue swollen, lacerated, and dotted with patches of grayish exudate; respiration normal; swallowing easy. The boy, although very hungry, was fed through a tube between the teeth. He at times made heroic but futile attempts to open his mouth and masticate solids. The opisthotonos was pronounced; the abdominal muscles were as hard as a board. In the arms and hands there were at times slight contractures; the thigh and leg muscles firmly contracted. By gentle manipulation, flexion, extension, and rotation of the legs could be executed; sensibility everywhere increased; knee-jerks active; Kernig's sign absent. Trousseau's sign absent; facialis sign impossible to elicit on account of the contracture of the face. The contractures in the extremities varied, sometimes almost completely disappearing; the masseters were always rigid; the teeth could never be separated more than one-sixteenth of an inch; the arching of the back would slightly increase or diminish, but never disappeared, day or night. The child was not nervous or emotional, but only

rough handling could accentuate the contractures. His voice was natural; urination and defecation in order; maculopapular rash present on trunk and extremities.

The idea of tetanus being paramount, ten doses of tetanus antitoxin were given September 12th to 21st. This treatment made the boy very restless, and had absolutely no effect upon the muscular rigidity.

From September 12th to 21st the child lay in the condition I have described—persistent trismus and opisthotonos and intermittent contractures of the extremities. The diphtheritic infection, proved by six positive cultures, caused no fever, prostration, or appreciable disturbance of the health.

September 21st the tetanus antitoxin was discontinued, and morphin, $^1/_{32}$ of a grain, was given hypodermically. A very comfortable night was the result, and, for the first time in three weeks, the opisthotonos disappeared, and the tight contracture of the masseters had somewhat relaxed. At my next visit the child was lying easily on his back, and could separate his teeth about one-quarter of an inch.

From September 21st to 27th the morphin, $^1/_{32}$ of a grain, was given every three hours, and the improvement was continuous. The patient slept on his back, with the muscles of the trunk and legs relaxed.

During the day there was slight arching of the back and contracture of the thigh muscles. The trismus was still present, but each day the jaws could be more widely opened.

September 24th to 27th the teeth could be separated one-half inch; there was still rigidity of the thigh and abdominal muscle; the back was normal.

September 27th. The patient sat up for the first time, and opened his mouth in a natural manner.

September 30th. The boy was able to walk and eat solids. From this date he rapidly improved, and was soon discharged from the hospital, with all muscular functions in order.

I have described a mild diphtheria associated with prolonged contracture of the masseter and dorsal muscles. From the third to the tenth day of illness, laryngismus and violent generalized muscular spasms occurred; after the tenth day the disease assumed a tranquil type, with persistent trismus and opisthotonos.

These contractures lasted twenty-one days, unaffected by tetanus antitoxin, but eventually relieved by morphin.

Trismus, opisthotonos, and muscular crises are symptoms absolutely foreign to diphtheria, and undoubtedly indicate the coexistence of another disease. The clinical picture would suggest tetanus of a most benign character. Tetanus is apt to be the result of an infected wound. The muscular crises and laryngospasms are increasingly severe, and usually end in death; while tetany is often allied with other maladies, and the motor symptoms are rather those of tonic contracture than recurring spasms.

The mixed infection of tetanus and diphtheria does not make the diphtheria milder, as was proven by the St. Louis epidemic of tetanus, in which horses immunized by diphtheria toxin afterward developed tetanus. Ten children given serum from these horses as a remedy against diphtheria, died of tetanus. The absence of Trousseau's and Chvostek's signs in my cases does not necessarily exclude tetany, as Escherich declares that in the type of tetany with persistent contractures these symptoms are seldom found, although similar groups of muscles are affected in true and false tetanus. The long-continued contracture of the jaw and dorsal muscles existing in a condition not dangerous to the patient is most suggestive of Escherich's pseudotetanus.

In concluding I would present some illustrative cases of pseudotetanus reported by Escherich and St. Ange Roger.

In 3 cases of Escherich's[4] the disease occurred in three boys, aged six, nine, and nine and a half years. The symptoms and course of the malady agreed in all the details. The children were previously healthy, with no hereditary taint, with no exciting cause; they first complained of stiffness in the legs, which hindered them from walking and compelled them to take to their beds. The rigidity rapidly spread upward and attacked the muscles of the back and jaw, so that the boys lay in an extended position, motionless and stiff as a board. The muscles of the entire trunk, neck, and legs were powerfully contracted; the rigid masseters gave the patients the grin of lockjaw. If the children were asleep or quiet the contractures lessened, but never disappeared; on the contrary, noises, handling, cold, or psychical emotion caused violent muscular crises, with opisthotonos and laryngismus. At the height of the disease these respiratory and muscular spasms came on spontaneously several times a day, and

were followed by profuse perspiration. In striking contrast to the woodeny appearance of the body, the arms, hands, and eye muscles remained freely movable. The disease increased in severity for a few days, remained stationary from three to six weeks, and then the contractures gradually lessened, and the boys, after considerable persuasion, were induced to use their legs again. After commencing to walk some stiffness remained for two to four weeks. Trousseau's and Erb's symptoms were absent. Escherich at first diagnosed hysteria, then idiopathic tetanus, finally deciding that the symptoms were a phase of tetany.

Baginsky[5] reports a case of tetanoid symptoms with diphtheria. The patient was a boy, aged four years and nine months. He complained of pain on chewing food, and was received into the hospital with characteristic trismus. Any efforts to open the mouth caused general convulsions, with arrest of breathing. On the edge of the tongue was a deep ulcer, covered with a grayish exudate. Considering the case one of lockjaw, Dr. Behring administered the newly discovered tetanus antitoxin for three days; no immediate effect was observed from the treatment, but the tetanic symptoms diminished in severity and disappeared in twelve days. The child afterward developed a suppurative arthritis, scarlet fever, diphtheritic ophthalmia, and diphtheritic paralysis; virulent diphtheria germs were found on the tongue, and the child infected two wards with diphtheria. No tetanus bacilli were found in the pus from the joints. The patient passed through this long series of illnesses in safety.

Question.—Was there a double infection of diphtheria and traumatic tetanus from a wound in the tongue, or were the tetanoid symptoms a rare complication of diphtheria?

Behring held that a double infection existed, and ascribed the muscular contractures and crises to tetanus.

Baginsky attributed the trismus and opisthotonos to a nervous complication of diphtheria, due to the proteus bacillus. Escherich called the condition pseudotetanus. Henoch declared he had never seen a similar case.

Guinon[6] reports a case of a boy, aged four and a half years, who was suddenly attacked with trismus, opisthotonos, and muscular spasms of the leg lasting thirteen days and subsiding with the appearance of a discharge from the ear.

Gomez[7] reports a case of a boy with grippe, complicated with

trismus and painful contractures of the arms and legs, so that he lay helpless in bed. Chvostek's and Erb's signs were present. The patient was relieved by saline infusions, although the contractures lasted a month.

Guinon[8] reports a case of a boy, aged four and a half years, who in the course of scarlatina was taken with stiffness of the legs, trismus, and contractures of the extremities. Trousseau's and the facialis symptoms were present. There were recurring muscular crises, with pronounced opisthotonos. The tonic muscular spasms were severe for eleven days, and had not entirely disappeared until the end of a month.

Cesare Cataneo[9] records a case of pseudotetanus in a boy, the symptoms subsiding abruptly after the evacuation of large quantities of lumbricoid worms. The child was of nervous heredity, good physique, save that he occasionally passed round worms. On May 2d, while taking a walk, he fell down, stiff and rigid, with the jaws tightly set. He was carried to the hospital, where the following conditions were noted: pronounced trismus, arms slightly contracted, legs rigid, pain in the back, recurring opisthotonos, frequent respiration, no fever.

The case was diagnosed as tetanus. Injections of carbolic acid were given. The trismus, opisthotonos, rigid leg muscles, and rapid respiration continued for eight days. The intelligence was clear, noise augmenting the muscular spasms. On the eighth day the boy was no better, and at the request of the father a large dose of castor-oil was given. A number of large, fetid stools containing masses of lumbricoid and pin worms was the result. The trismus and opisthotonos immediately disappeared, and were obviously due to tetany caused by intestinal toxemia. A long, serious attack of bronchopneumonia followed. There was no recurrence of the tetany, and the child ultimately recovered.

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DISCUSSION.

DR. JACOBI.—I have listened attentively to the history and think in all its phases it is a case of mild tetanus. We see a great many of these cases getting well and I do not see why we should adopt a different name just because it looks a little different from some others. Morphin and chloral hydrate have certainly cured and relieved a great many of these cases and I see no reason why we should now refer to them by a different name. Escherich differs from many of us in his ideas as to their connection with rachitis. I do not believe we should follow him in a new method of nomenclature.

DR. KOPLIK.—I wish to hear the experience of other members of the Society as to tetany being rare. A year scarcely passes but that I see several cases. In a large ambulatory clinic it is common every spring to have these cases come in. The reason that Escherich has seen so many cases is that in his wards there have been epidemics of tetany. In my hospital ward I see cases of tetany quite frequently.

DR. HOLT.—I would like to endorse what the last two speakers have said. I see 3 or 4 cases of tetany every year; and, further, I believe in what Dr. Jacobi has said, that it is a mistake to have the opinion go out that tetany is not connected with rachitis. The evidence seems to me convincing that it is the underlying cause and that intestinal toxemia is usually the exciting cause. The history of the case reported appears to me like that of a case of mild tetanus.

DR. JACOBI.—I would like to appeal to the older gentlemen here and those who have followed the history of medicine in America for the period of a few dozen years. There was a time when we did not see tetany and did not know from personal experience what it was, because we did not know then what rachitis was. We have seen it increase from year to year in the last forty years, with the increase of poverty, bad food and bad air. We see rachitis more and more from year to year. As late as thirty years ago I published for the first time observations on craniotabes, and there was only one man who had preceded me, a few months only, viz., my friend Parry, of Philadelphia. Now rachitis is prevalent and we see tetany. There is hardly a month that a case does not turn up. How many has Dr. Huber seen?

DR. HUBER.—Five or six a year.

DR. JACOBI.—Every one who has dispensary work sees them and frequently they turn up in our private practice, and, as poverty increases we shall see more of them.

DR. KERLEY.—I see 5 or 6 cases a year in the out-patient

work and I associate it with rachitis and intestinal infection. I never saw a case where the intestine was in a normal condition.

DR. GRIFFITH.—Some years ago I had occasion to study tetany as it occurred in America and published the results. This paper has sometimes been quoted to show the rarity of the disease in this country. It is, however, hardly a proof of this, but rather that the condition was either seldom recognized, or, if recognized, was not reported. Apparently there is no doubt that the disease has been uncommon here as compared with its frequency in parts of Europe. This seems to depend upon the fact that rickets was not a common disease in America twenty years and less ago. At that time medical graduates scarcely knew what rickets was, and probably most of them had never seen a case. Now instances are so common that the medical student is tired of seeing them. With this increase in rickets has come the increase in the number of cases of tetany.

With regard to the interesting case of Dr. Snow's, I am inclined to share the feeling of others that it would be difficult to distinguish it from tetanus.

DR. SNOW.—It is very evident that tetany is geographical in its distribution, for it was frequent at one time in parts from which it has now disappeared. As to whether this was tetanus or a form of tetany is a problem. I see tetanus, of course, in the child wards and use morphin and chloral, but this case did not suggest tetanus when I first saw it. The subject has been much more studied in Austria and by Escherich than in this country. I certainly stand by my position that this was not a case of tetanus.

ADDENDA.

PROCEEDINGS OF THE THIRTEENTH SESSION.

CONGENITAL ABSENCE OF THE ABDOMINAL MUSCLES, WITH DISTENDED AND HYPERTROPHIED URINARY BLADDER.

BY WILLIAM OSLER, M.D.,

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In the summer of 1897 a case of remarkable distension of the abdomen was admitted to the wards, with greatly distended bladder, and on my return in September, Dr. Futcher, knowing that I would be interested in it, sent for the child. The accompanying Figures, I. and II., from photographs, show a very remarkable and unusual pattern of "abdominal tumidity," differing in an interesting way from the picture of the dilated colon in children, and resembling rather that of the ascitic abdomen.

The examination showed that the child had practically no abdominal muscles.

On looking up the literature I can find reports of only 2 similar cases. In the Clinical Society's Transactions (Vol. xxviii., 1895), R. W. Parker describes the condition of a newly-born infant, weighing five and a half pounds, with a very large, flaccid abdomen, through which the outlines of the intestinal coils could be clearly seen, and the outlines of the abdominal organs easily felt. The abdominal wall was as thin as parchment. Along the middle line, where the rectus muscles should be found, there was little more resistance than over the lateral regions. The oblique and transversalis muscles were apparently quite undeveloped. The umbilicus was not depressed, it was in normal position, but resembled a surface scar. The child died not long after birth. There was no trace of any muscle representing the transversalis abdominis. There was a thin layer of muscular fibres passing from the cartilages of the ribs to the level of the eighth costal cartilage, where there was the first linea transversa. The body of the muscle was well marked on the right, but on the left it was but faintly seen. Further down there was the merest trace of muscular fibres, representing the rectus on either side. The most remarkable associated condition in this case was the enor-

mous hypertrophy of the bladder, which was situated wholly within the abdominal cavity. There was no obstruction anywhere in the urethra or prepuce. The openings of the ureters into the bladder were quite free. The ureters and pelves of the kidneys were greatly dilated and hypertrophied.

FIG. I.
From the Johns Hopkins Hospital Bulletin.

In 1896, Dr. Leonard Guthrie reported to the Pathological Society of London (Transactions, Vol. xlvii.), the history of a male infant, aged nine weeks, pigeon-breasted, very bony and emaciated, with a greatly distended abdomen. Extending between the pubes and the white, linear cicatrix corresponding to the umbilical scar there was a smooth, elastic tumor, corresponding to a distended gall-bladder. The abdominal walls were excessively thin and loose, and seemed to show the coils of the distended intestines on either side, but postmortem these coils which looked like the intestines proved to be the enormously dilated and convoluted ureters. The liver, spleen and kidneys could be easily palpated. The child wasted rapidly and died when about ten weeks old. Of the recti only the two upper segments

as far as the second linea transversa showed muscular fibres. Below this level no trace of muscle could be discerned. The costal origins of the obliqui and transversales showed muscular structures for about two fingers' breadth below the ribs. The muscles of the back, of the thorax and of the extremities were well developed. Here again the most remarkable features related to the urinary organs. The bladder reached as high as the scar of the navel, and the walls were a quarter of an inch in thickness. The ureters were dilated to the size of the small intestines of an adult, and were remarkably tortuous. After death they exactly resembled, and at first were taken to be, portions of distended small intestine, as they were thought to be when seen through the weakened abdominal walls during life. The orifices of the ureters into the bladder admitted a blow-pipe. There was no obstruction in the ureters; there was no stricture of the urethra, and no phimosis. The kidneys were not enlarged, but the pelves were dilated. The position of the testes was not stated.

FIG. II.
From the Johns Hopkins Hospital Bulletin.

An important point in Dr. Guthrie's case was that there was

no trace of a urachus, and the bladder was closely adherent to the inner surface of the umbilical scar, so much so that it could not be removed without the scar and the adjoining portions of the abdominal skin.

THE HISTORY of my case is as follows:

Claudius K., aged six, admitted July 13, 1897, complaining of stomach trouble, and difficulty in passing the urine. The chest has been deformed, the mother says, since birth.

The family history is good. One other child; well and strong; parents are healthy.

PERSONAL HISTORY.—The child was well until the second summer, when he had severe stomach trouble. There have been recurrences of these attacks each year. From the account some of them have been gastric attacks, with nausea and vomiting, but others, and apparently the chief troubles, have been with the urine. The spells last four or five weeks, and they have been getting more frequent. In the intervals he is pretty well and strong, and has a large appetite.

His present attack began about a week ago, and he complained of pains in the abdomen and much burning sensation in passing water. He has become very weak; has not had any vomiting. He has had some headache.

The patient was a poorly nourished child, looking anemic. He complained of much pain, chiefly in the hypogastric and lower umbilical regions. On inspection the condition to be described was noted by Dr. Futcher, but in particular there was a remarkable fulness in the hypogastric and lower umbilical regions, which were occupied by an ovoid mass corresponding to a dilated bladder. The urine which was obtained by catheter was free from albumin, contained a good many leukocytes. The child had a temperature ranging from 99° to 102°. He passed the urine very frequently, an average of from 60 to 70 cc. In the twenty-four hours ending 5.30 on July 13th he passed urine 20 times, a total amount of 1090 cc.; on the 14th he passed urine 18 times, a total amount of 835 cc.; on the 15th he passed urine 15 times, a total of 1060 cc.

The condition was so unusual that on my return in September the case was sent for, and on the 8th I dictated the following note:

In the erect posture the attitude is very remarkable. It is not

quite symmetrical, being fuller on the right side than on the left. The navel looks stretched and distended. It is linear, forming a furrow about an inch in length, and below it are furrows in the skin—crow's feet. Above there is seen distinctly on either side the attachment of the recti to the sternum and costal margin. The skin over the abdomen is thin; the veins are a little prominent. When he bends back slight movements of the abdominal muscles beneath the skin are seen. (See Fig. I.)

RECUMBENT.—Belly flattens out in front, extends at the flanks. Coils of intestines can be seen in peristalsis. Extreme relaxation of abdominal walls; no resistance; fingers can be passed everywhere to the spine. Three fingers can be passed under costal margin over liver nearly 6 cm. The edge of the liver can be felt in its whole extent, and the fingers can be thrust almost as far under it. The bladder could be felt as a firm ovoid body, reaching almost to the navel.

Spleen can be felt on deep pressure. Both kidneys can be felt.

He cannot raise himself off the bed without turning over. As he makes the attempt the abdomen is thrust forward and slight contraction is seen of the expanded abdominal muscles and recti.

The deformity of the thorax is very remarkable. Harrison's grooves are unusually marked, corresponding to the sixth costal cartilage. The lower portion of sternum is thrust forward, forming almost a right angle with the xiphoid cartilage. As shown in the photograph it is remarkably prominent, and is fully 3 cm. above the level of the skin in the intercostal furrows. (See Fig. II.)

There is a condition of cryptorchidismus. The testes are not to be felt in the groins.

REMARKS.—These cases illustrate a very remarkable form of congenital defect. The deficiency in the abdominal muscles, and the high position of the bladder are associated conditions due to arrest of development. We could not say definitely in my case whether the bladder was adherent to the umbilical scar. Dr. Guthrie regarded the hypertrophy of the bladder and the dilatation of the ureters as secondary, due to the fact that in his case, being firmly connected with the umbilical scar, it was unable to contract downward and to empty itself completely. In its effort to do so it became hypertrophied and dilated, and the accumulation of urine caused backward pressure and dilatation of ureters.

In reply to a question, Dr. Bardeen, one of Professor Mall's associates in the Anatomical Laboratory of the Johns Hopkins University, who has been specially engaged in a study upon the development of the muscles, writes as follows: "Two possibilities suggest themselves to me in the case:

"(1) It is possible that the lack of resistance normally met with in the abdominal wall by the bladder at the time the kidneys begin to secrete urine may cause the bladder to expand rather than to empty secretions into the amniotic cavity through the urethra.

"(2) Under normal conditions the growth of the abdominal musculature into the *membrana reuniens,* the early covering of the abdominal cavity, is preceded by the formation of a vascular plexus supplied from above by the internal mammary, from below by the epigastric artery. It is possible that an abnormal arrangement of the blood vessels in the embryo prevented the formation of this plexus, and impeded the growth of the abdominal musculature, and that at the same time circulating disturbances gave rise to the abnormal conditions found in the bladder and ureters."

THE PLACE OF CEREALS IN INFANT FEEDING.

BY HENRY DWIGHT CHAPIN, M.D.,

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A discussion of the propriety of using cereals in the feeding of infants may be approached from a theoretical or a practical and clinical standpoint. It is believed that the profession has recognized by long clinical experience that a certain judicious use of the cereals is frequently beneficial. On the theoretical side, it must be confessed that, at first sight, the employment of a material in a form not found in human milk may appear unwarranted to those who desire a strictly scientific reason for all procedures. A discussion of this question from the latter standpoint may be of interest if we bear in mind always the character and composition of cow's milk, with which the physician has to deal in his home modification to suit the infant's digestion.

The question then at issue is whether the use of cereals with cow's milk in infant feeding is unnecessary and unscientific. Unnecessary, because of the claim that cow's milk when diluted with water gives as fine, if not finer, curds than when diluted with cereal waters; unscientific, because nature is not being followed.[1]

The claim that cereal waters have no more effect on the curd of cow's milk than plain water has been abandoned, as it was based on the precipitation of casein with dilute acids and not upon its coagulation with rennet, which is what takes place in the infant's stomach.

When it comes to preparing cow's milk for the infant there arises the question, what constitutes following nature?

It is often claimed that the difference between woman's milk and cow's milk lies in the different percentages of fat, sugar, proteids, ash and water present, and that to follow nature the percentage of these ingredients of the infant's food should be made the same as those of average woman's milk and varied to suit the infant's digestion.

To determine the composition of a specimen of milk, resort to a chemical analysis is had. A portion of the milk is weighed and evaporated to dryness; the residue is weighed and called total solids;[2] the loss in weight is called water.[2] A part of the total solids is ignited and weighed as ash or salts.[2] What ether will extract from another part of the total solids is weighed and called fat.[2, 3] Another specimen of the milk is put through a complicated process to convert all the nitrogen present into the form of ammonia. The ammonia is distilled off and determined. The weight of the nitrogen in the ammonia is calculated and multiplied by 6.25 and called proteids, as 100 part of proteid bodies contain on an average 16 per cent. of nitrogen which multiplied by 6.25 equals 100.[2, 3, 9]

The weights of the ash, ether extract, and nitrogen multiplied by 6.25 are added together and subtracted from the weight of the total solids, and the difference called sugar;[4] or a specimen of the milk is precipitated with various reagents, such as acid mercuric nitrate or copper sulphate, filtered, and the sugar in the filtrate determined by reduction of alkaline copper solution, or by the rotation of a ray of polarized light.[2, 3] When the sugar in woman's milk is determined by both reduction of alkaline copper solution and rotation of a ray of polarized light, there is a difference in the quantity present, which indicates two sugars.[2] Direct separation of the sugar of cow's milk shows two carbohydrates to be present,[5, 6, 9] but no method of quantitatively separating the sugar of milk in a state of purity is known.[2]

As the proteids of meat, milk, cheese, eggs, cereals, and even fertilizers are all determined by multiplying the weight of the nitrogen present by 6.25, or some other factor,[3, 9] and as this gives no clue to the properties of the proteids, chemists divide them into two great classes:[9]

(1) Native proteids, those that can be separated from animal fluids and tissues by chemical means without losing their identity.

(2) Modified proteids, those that are derived from native proteids by the action of heat in coagulating; by the action of acids or alkalies in forming acids or alkali albuminates, or by the action of proteolytic enzymes in forming albumoses and peptones.[9]

In milk we have to deal with native proteids and these are divided into groups according to their solubilities, as: albumins,

soluble in water; globulins, insoluble in water but soluble in dilute neutral salt solutions or weak acid or alkali solutions; and neucleo-albumins, which are soluble in dilute alkali solutions. There are no sharply dividing lines between these groups. The neucleo-albumins differ from the albumins and globulins in containing phosphorous. There are a number of kinds of albumins, globulins and neucleo-albumins.[9]

In both woman's and cow's milk are found albumin, globulin, and nucleo-albumin (casein). The albumin in woman's milk forms from one-third (Richmond) to two-thirds (Koenig) of the total proteids, while in cow's milk the neucleo-albumin (casein) forms about four-fifths of the total proteids. There seem to be some soluble proteids similar to albumin in every kind of milk that has been examined.

When the nitrogen of the albumin, globulin, and neucleo-albumin (casein) of milk is determined separately, there is found to be less nitrogen than when the nitrogen is determined directly from the milk; one-eleventh of the nitrogen of woman's milk and one-sixteenth of the nitrogen of cow's milk is thus unaccounted for.[9]

Backhaus and W. Cronheim recently reported the analyses of twelve samples of human milk. In agreement with the results of Camerer and Söldner, "it was found that there was a discrepancy between the total solids and the sum of the constituents of the total solids, as determined separately, amounting to from 0.68 to 2.045 per cent. This is held to represent an unknown constituent which passes into the filtrate when the albuminoids are precipitated with alcohol."[7]

When milk is taken into the stomach it is first clotted by the rennet which produces a chemical change in the casein. A clotted jelly which encloses the fat is formed from cow's milk,[8] and a soluble proteid known as "whey proteid,"[9] similar to an albumose is split off from the casein. A clear fluid, known as whey exudes from the curds and contains the "whey proteid," albumin, globulin, and sugars of the milk. To quote J. Reynolds Green,[10] "When casein either in milk, or in solution in dilute alkalies, is acted upon by rennet in the presence of a small quantity of calcium phosphate, it is at once chemically altered. It gives rise to the formation of a body which has been named tyrein, which is the proteid constituent of the clot. When the whey has separated out, it is found

to contain a by-product having properties resembling those of a soluble albumin, except that it is not made insoluble by boiling. It is consequently distinct from the serum albumin, or lactalbumin, present in normal milk. The formation of this by-product, when solutions of pure casein are treated with rennet, shows that it results from the alteration of the casein. The clotting of milk by rennet is consequently not to be confused with the precipitation of casein by acid."

The curding process consists essentially of a separation of the fat and casein from the easily absorbable proteids and carbohydrates.

Albumin, globulin and albumose can be absorbed with little digestive effort. Neucleo-albumin (casein) must be peptonized before absorption. All proteids are in some unknown way changed before passing into the blood, for even peptone is excreted by the kidneys when injected into the blood.[9]

During digestion there is an increased elimination of nitrogen which is proportional to the intensity of digestive work, and an increase of about 15 per cent. in the quantity of oxygen consumed and a larger increase in quantity of carbon dioxid thrown off.[11] This shows an immediate demand for proteids and carbohydrates at the beginning of a meal; it has just been shown that the first step in the digestion of milk is a separation of easily absorbable proteids and carbohydrates (sugars) from the casein and fat which require digestion as they are left in a semi-solid state.

The character of the curd that is formed in the stomach seems to depend on the milk that is used.[2, 10]

The milk of the cow, goat, sheep and buffalo, which are ruminants, shows a wonderfully close resemblance in chemical composition, and the curds formed with rennet are solid.

The milk of the mare, mule,* and ass, which are not ruminants, also shows close resemblance in chemical composition, but is quite different from that of ruminants, and the curds formed with rennet are soft and flocculent. The milk of the cat and dog, carnivorous animals, is very much alike in composition, but decidedly different from that of either class of herbivorous animals. Nothing is known about the curding properties.

* It may be of interest to note that the female mule will sometimes reproduce by union with either the horse or the ass.

Among the herbivoræ, at least, the rule seems to be: animals that ruminate, furnish their young with milk that curds in solid lumps, and animals that chew their food before swallowing it, furnish their young with milk that curds in loose flakes. The human stomach receives food in a finely divided state and woman's milk curds in loose flakes.

A glance at the following table constructed from analyses in Richmond's "Dairy Chemistry"[2] will also show that the milks that form solid curds with rennet are furnished by animals whose normal digestion is prolonged and whose intestinal canals are relatively very much longer than those of animals whose milk forms soft flaky curds, and that carnivorous animals, whose intestines are relatively very short, furnish milk rich in proteids, half of which are readily absorbable. The gastric juice of a dog will digest in the same time four times as much cooked albumin as that of a sheep.[12]

It is also interesting to note the rapid growth of animals whose milk is rich in proteids, much of which is present in a soluble form.

	Water.	Fat.	Sug.	Cas'n.	Alb'n.	Ash.	Curds.	[12] Ratio of intestine to body (length)	[13] Doubles weight in
Carnivorous :									
Cat,	81.63	3.33	4.91	3.12	5.96	.58	?	4 to 1	9½ days.
Dog,	75.44	9.57	3.09	6.10	5.05	.75	?	6 to 1	8 days.
Ruminant :									
Ewe,	79.46	8.63	4.28	5.23	1.45	.90	Solid,	27 to 1	10 days.
Goat,	86.04	4.63	4.22	3.49	.86	.76	Solid,	27 to 1	19 days.
Buffalo,	82.63	7.61	4.72	3.54	.60	.90	Solid,	.....	.....
Cow,	87.10	3.90	4.75	3.00	.40	.75	Solid,	20 to 1	47 days.
Non-Ruminant :									
Mare,	89.80	1.17	6.89	1.84		.30	Flocln't,	12 to 1	60 days.
Mule,	91.50	1.59	4.80	1.64		.38	Flocln't,	11 to 1	.....
Ass,	90.12	1.26	6.50	1.32	.34	.46	Flocln't,	11 to 1	.....
Human :									
Woman,	88.2	3.30	6.80	1.00	.50	.20	Flocln't,	6 to 1	180 days.

A table essentially the same was also constructed from analyses by Koenig. The percentages were slightly different, but not more so than would be accounted for by variations in milk.

During the first days of life, all young suckling animals are furnished colostrum, which is rich in proteids consisting largely of albumin and globulin,[2, 5, 6, 14] the most easily absorbable forms of native proteids, and sugar, which, in the case of the cow, is largely dextrose,[2, 5, 6, 14] the sugar found in the blood. Colostrum

will hardly curdle with rennet,[2, 6, 14] and the stomach of a calf under three days old furnishes a very weak rennet.[8]

The change from colostrum to milk, which takes place during the first week or two after birth, consists of a reduction of the easily absorbable proteids and sugar, until in normal cow's milk, the easily absorbable proteid is less than the casein,[2] which calls forth considerable digestive effort. All normal milk contains a portion of the proteids in a form that is not acted upon by rennet. Not much is known about the sugars, as they are estimated together, and then not directly.[2]

When mixed solid food is taken by a child or an adult, it must be chewed and mixed with saliva, by which some of the starch is converted into dextrin and maltose, which are easily absorbed.[9] Thus in every stage of life the food is adapted to the condition of the animal. At birth when the digestive process is being set up, the food requires little digestion; as the animal's digestive apparatus commences to work, the food in the stomach gradually changes from a fluid to a semi-solid state peculiar to each class of animals. The increased demand for proteids and carbohydrates during digestion is met by a separation of the soluble proteids and carbohydrates as soon as the milk is swallowed, and when solid food is taken, a supply of soluble absorbable carbohydrates is produced by the action of saliva on the starch. Furthermore, the secretion of all the digestive juices is promoted by the absorption of nutriment at the beginning of a meal.[10]

When cow's milk diluted with water is given to an infant the quantity of readily absorbable food is reduced to almost nothing, and the proteids will form a solid curd if the conditions are right. In practice, with all the advantages of a laboratory and an almost perfect milk supply, it is found necessary to begin an infant on proteids one-eighth to one-fourth of the quantity found in woman's colostrum and milk, and gradually increase the quantity as the infant can digest them. The one element that is kept up is sugar. This practice conforms to observations that have shown that an amount of ingested proteids smaller than the amount that is being excreted may enable an animal to show a gain in proteids if additional carbohydrates are furnished at the same time, and that increased quantities of carbohydrates in the food decrease the proteid metabolism more regularly and constantly than increased quantities of fat.[9]

The practice of the advocates of percentage feeding shows that an adjustment of the percentages of fat (ether extract), proteid (nitrogen x 6.25), and sugar of cow's milk, to equal those of woman's milk does not enable them to strictly follow nature in percentages, for nature most decidedly protests when the same percentage of proteids of cow's milk as is found in woman's milk is given to a young infant; this shows that percentage feeding simply records the quantities of these ingredients of *cow's milk* a particular infant is able to digest. Good clinical results have been obtained, and so they have been with other methods, but the claim that in this way only nature is being followed is not well founded. The introduction of system into infant feeding has been a great advance and has come to stay, but it has emphasized the fact that changing percentages does not change cow's milk into woman's milk.

Practice has shown that each infant is a law unto itself, no matter what method of feeding is employed. Now, if nature is to be followed, the infant's food should curd in flocks or loose masses and there should be a moderate supply of easily absorbable food separated at the time the curds form. As sugars have such a sparing effect on proteids they can be used to replace part of the soluble proteid which is reduced in quantity by dilution of cow's milk.

When sugars of milk are examined directly and not determined by "difference," sugar of woman's milk is found to be different from sugar of cow's milk[2] and cow's milk is found to contain two carbohydrates, one of which Béchamp considers to be dextrin.[9] Chemistry shows cane sugar, milk sugar and maltose to have the same composition (C_{12} H_{22} O_{11}) and all three rotate a ray of polarized light in the same direction (Cane Sugar, +66.5°, milk sugar, +52.5°, maltose, +137°). Cane sugar and milk sugar when injected into the blood are excreted unchanged. Moderate quantities of dextrin and maltose when injected into the blood are retained.[9, 10] The first products of digestion in the adult are dextrin and maltose, and these have been proved to be absorbed from the stomach and to provoke the secretion of all the digestive juices.[10] Even young infants can digest small quantities of starch.

In 1893 it was discovered that there were two enzymes, diastase and glucose, in the blood serum and lymph, one of which converted glycogen, which is the form in which carbohydrates are stored in the liver, into dextrin and maltose, and the other which con-

verted maltose into dextrose, which is the sugar found in the blood, and which is converted again into glycogen in the muscles and cells of even the embryo;[9] so it cannot be claimed that in the addition of small quantities of starch, dextrin or maltose to cow's milk elements are added than an infant is unprepared to care for.

To break up the curd of cow's milk and furnish a small quantity of easily absorbable food, cereal gruels, in which the starch has been converted into dextrin and maltose, are the most practical and desirable agents. It has been denied that cereals or any kind of mechanical attenuant had any effect on the curd of cow's milk,[1, 15] but this was found to be based on a mistaken conception of the curdling properties of milk. Recently it has been admitted that cereals gave the finest curds of any diluent,[11, 5, 16] but it was claimed that all the effect of the cereal is lost when the starch is digested, especially if the digestive ferment is active.[16] How much effect a digested gruel has on the curding of milk depends on the strength of the gruel and the dilution of the milk. The very best effect as far as digestive effort is concerned is obtained when the starch is completely gotten into soluble forms so that the particles of proteids and cellulose of the cereals are free. Any one who has seen junket made has noticed how the curd shrinks away from the dish. The curd of cow's milk which has been diluted with thoroughly digested gruel shrinks away from each little particle of cellulose and proteid of the cereal, and allows a rapid exudation of the soluble, easily absorbable portion of the food consisting of albumin, globulin, "whey proteid," dextrin, maltose, and milk sugar. Sometimes the curds may hold their form, but they are spongy and soft, never leathery, and break apart on slight agitation.

To find what strength of the digested gruel was needed to prevent the formation of leathery curds, three heaping tablespoonfuls of wheat flour, weighing 1,562 grains, were cooked in a double boiler for two hours with three pints of water in my kitchen; as it could be done in any household. This gruel was cooled so that it could be tasted (130° F.) and one teaspoonful of cereo, a glycerite of diastase, was added. At the end of ten minutes iodin failed to show starch.

A quart of milk was obtained and tested for fat and showed 4 per cent. It was amphoteric to litmus paper, but showed 0.15 per cent. acidity to phenolphthalein. The acidity of milk is never de-

termined directly,[2] but by neutralizing with alkali, the end of the reaction being indicated by the change of some color indicator. Milk is nearly twice as acid to phenolphthalein as to litmus.[2]

One part of this milk was diluted with an equal quantity of water, another part with 1½ per cent. starch jelly and another with the digested gruel; 0.15 per cent. HCl and some liquid rennet were added to each.

The artificial curding of milk with rennet is dependent on the temperature acidity of the milk and the strength of the rennet;[6, 8, 14] whether the acid and the rennet are added together or separately, and if separately which first.[8] The curd produced when rennet is first added is tougher than when acid is first added, as the rennet has then more opportunity to come in contact with all the casein, for the acid has a tendency to throw the casein out of solution before the rennet can come in contact with it.[8]

In making these tests it was desired to make the curds as tough and leathery as possible, so the rennet was first added to the cold diluted milk and the acid afterward. The mixtures were then warmed without agitation and allowed to shrink until the curd of the specimen diluted with water could be dropped on the floor or tossed like a ball without breaking. This was the severest test that could be devised.

The curds of the specimen prepared with 1½ per cent. starch jelly and the digested gruel were very much alike in texture. Neither was tough and leathery. This difference was noticed; in one the curds of the milk were coated with starch, in the other the curds were thoroughly exposed.

It is not necessary to use a digested gruel stronger than one heaping tablespoonful of flour to the pint for any dilution of milk.

If when modified milk disagreed all milk was cut off for a few feedings, and then a new beginning made, a great deal less trouble would be experienced than by attempting to shift percentages slightly. It might be found that the milk itself was at fault. Something has to be fed when milk disagrees. The question is, shall the physician furnish this substitute or turn the baby over to the manufacturers of proprietary infant foods?

There is no form of nourishment so easily borne as predigested cereal gruels, and when properly used these are of great benefit, even when the infant is breast-fed.

A glance over the standard text-books and recent articles[17—22]

will show how often the cereals in one form or another are called into use; almost always when milk must be withheld; and it is just at these times that infant foods, which are made principally of cereals, are used.

If instead of claiming that it is unscientific to give a baby anything but milk, the great advantage of temporarily withholding milk when it disagrees, and feeding cereal gruels instead of proprietary foods was taught, and then to return to milk feedings by gradually adding milk to these gruels there would be less recourse to proprietary foods and more practitioners would learn to become scientific modifiers of milk. This method can be followed anywhere and in no way interferes with the principle of shifting the percentages of fat, sugar or proteids to suit the infant's digestion. In fact, this is exactly what should be done in every case.

It would seem that by beginning with a predigested cereal and adding milk to this, nature would be pretty closely followed in furnishing part of the food in a readily absorbable condition and in rendering the remaining portion soft and flocculent. This is practical and seems more scientific than making the standard what ether will extract from the food, the amount of nitrogen it contains, and the rotation of a ray of light or the reduction of alkaline copper solution it produces.

The conclusions are, that chemical analyses of milk are not the only scientific bases of comparison; that nature adapts an animal's milk food to its digestive system, and that cow's milk and woman's milk were intended for different digestive systems; that as cow's milk forms solid curds and woman's milk flocculent curds, the curd of cow's milk intended for an infant should be broken up mechanically; that as cereal gruels mechanically break up the curds of cow's milk, and as infants are able to utilize them, their use is rational.

It is often preferable to make the standard diluent of digested gruels, as they not only break up the curds but expose a surface of milk proteids and not starch, and furnish a certain amount of nourishment that is immediately available for carrying on the work of digestion, taking the place of part of the soluble proteids, and also form a satisfactory substitute for milk when it must be withheld for a few feedings. A great variety of food can be supplied an infant by means of digested gruels at a trifling expense, and the tendency is always to get back to milk feedings and not to

keep on indefinitely with a diet of carbohydrates, as when most infant foods are used.

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DISCUSSION.

DR. ROTCH.—I would say that I think there is a mistake in the chart in the figures relating to woman's milk. Dr. Chapin states that the casein, by which I think he means caseinogen, is greater than the amount of albumin in woman's milk. To say that there is twice as much caseinogen in human proteids as there is of the albumins I should doubt very much. Such a statement is contrary to all the analyses that have been made and I think it must be a mistake in the copying, for Dr. Chapin tells me that his figures were taken from Richmond's work. Now Richmond's figures differ entirely from what we consider at Harvard to be the proper ones, Koenig's as quoted by Hammersten. We shall have to investigate this matter again and, perhaps I am wrong, but I am pretty sure that the figures I have been using are correct.

I should like to say in regard to the cereals that this subject has been discussed a great deal regarding the point as to whether the coagulum was finer and smaller when plain water was used than barley water. I think there is no question but that Dr. Chapin is right about this as it has been proven that the coagulum is finer with barley water provided the latter is of the right percentage, that is, about .7 of starch. If the percentage is less than this the coagulum is not so fine and when it is greater it is apt to cause indigestion. I have said this because I had taken the opposite ground before. I know now that I was wrong because our experiments at the Massachusetts' General Hospital as well as those of Dr. Chapin have shown that he was right and I was wrong. In regard to the necessity of using them, however, I hold to the same opinion that I did before, namely, that there is no necessity for using a starch to break up the curds.

The paper is exceedingly valuable but I believe that you can use the whey albumins in many cases with benefit. If, however, it is proved that in splitting up the proteids you get something that is poisonous as a food then they should not be used. I believe, however, that this has not been proven yet. I have used this method of splitting the proteids in feeding delicate babies continuously since last July and the results are more satisfactory than when I prescribed the total proteids. If you use the whey proteids you can get a mixture more nearly approaching human milk than if you base your calculation upon the total proteids. Furthermore, the coagulum that is obtained in this way is as fine as that obtained with barley water and hence there is no use in using barley water even to break up the curd.

The author of the paper could not have meant exactly what he said, when he spoke of its being better to use cereals because if you did not the people would use the patented foods. The question is are these things good for the infants or are they not. To say that if you do not give them starch, somebody else will is not the proper way to approach this question. It would be playing into the patent food men's hands to take the position which I understood him to take.

DR. KOPLIK.—During the past two years I have made an extensive trial of dextrinized gruel food. We should distinguish clearly between the healthy and the sick infant in considering this subject. In my experience healthy infants do quite well on ordinary modifications of milk without the addition of cereals, dextrinized or otherwise. With the sick infants we must sharply define the acutely, from the chronically, ill. In the former class the milk food is stopped and when symptoms abate I have found it advisable in some cases to return to dextrinized gruels before placing the patient on ordinary milk dilutions. I do this even though there is still some diarrhea. Taking up the infants who

suffer from chronic intestinal derangement we have an entirely different problem to deal with. Keller has shown that in these cases there is an acid intoxication of the system of intestinal origin. Milk mixtures prepared in the ordinary way perpetuate the faulty conditions in the gut. By the administration of dextrinized gruels containing malt sugar a certain proportion of these cases not only improve but the improvement, and increase of weight, is progressive. There is something more in the improved assimilation of the milk of these mixtures than the breaking up of the casein curds. The addition of dextrinizing agents to the milk, omitting the cereals, does not increase the weight of the infants.

DR. ROTCH.—I should like to say that some very careful work has been done at the Massachusetts' Hospital in the dextrinizing of starch by Dr. Franklin White, and that his results are not at all the same as those obtained by Drs. Koplik, Chapin and Keller. Dr. Koplik assumes that we do not go so far as Keller and study what is going on in the intestine as well as studying the composition of the milk. We have taken all that into consideration and I think I may fairly say that Keller does not know anything about this new method of feeding. I am sure that Dr. Koplik himself has not made a proper trial of this method of splitting up the proteids.

DR. GRIFFITH.—Listening as I have year after year to these interesting and animated discussions on the feeding of infants, listening in a manner as an outsider who has no particular theory which he is anxious to champion, not that I have not some very decided views, but that I do not intend to advance them on the present occasion, I have been impressed with two facts in particular. The first is that no one of the speakers is entirely right in this matter; and the other, that no one is altogether wrong. And I believe, gentlemen, that from the nature of things and the conditions attached to this subject, that this must ever be the case. Sometime ago I read an interesting discussion in one of the German medical journals, in which the speaker was advocating the addition of egg-white to the milk mixture. A well-known authority in discussing the subject answered: *"Die Frau ist doch kein Huhn!"* Now this is quite true, and the addition of white of egg can never make a milk which is like human milk. Yet, on the other hand, I have very frequently found it exceedingly useful in the feeding of infants, as have many other physicians. As regards its use there is, then, no absolutely right and no absolutely wrong.

The same remark might have been made by the speaker about cow's milk: *"Die Frau ist doch keine Kuh!"* In cow's milk we are dealing with a substance which you can never make just like human milk. It is beyond the possible. Therefore, there can be

no absolutely right way in which we must treat it before it is given to a child, since, after all, it is an unnatural food, and any mixtures made with it are at best but approximations to, and imitations of, the natural article.

In the first place then, we are dealing with an unnatural milk, and no absolute rule can apply to it. A second reason why there is no rule of feeding which can be called absolutely right is that we are dealing with babies as individuals. This fact entirely precludes the following of any rigid rule. The man who tries to do it does not at all grasp the conditions which exist. A third, and very important, factor in this question is the individuality of the man who has the case in charge. One physician uses certain kinds of feeding, learns what he can do in this way and how far he can depend on his methods. His results are good. Another physician, equally competent, follows another plan and gets equally good results. He learns how to use his methods, as a workman learns to use his tools. No one is quite right, and no one quite wrong. Just as we cannot condemn a colleague who gets good results with a certain drug on the ground that we use some other drug in getting the same results.

I may by some be considered heretical in regard to the scientific modification of milk. I am not so, but am a firm believer in the use of this method of feeding. I prefer it to any other for nearly all cases. But I think we must never forget the impossibility under the circumstances of invariably applying absolute scientific principles to infants, who wont respond in a scientific manner, when, too, we have not scientifically accurate substances to work with, and are as yet far from understanding all there is to be learned in the science of infant feeding.

DR. HOLT.—I agree very nearly with what Dr. Koplik said in the beginning of his remarks with reference to the confusion that has arisen from considering two or three matters at the same time. One proposition that should be brought out is whether it is better to put dextrinized gruels in the food for all children, or only when certain disorders of digestion exist. From the first the proposition I should certainly dissent, but with the second I concur though I have arrived at my conclusion from somewhat different reasons from some of those given. I quite agree with Dr. Griffith that one gets the best results with the tools with which he is most familiar. If he is an intelligent man and watches his babies he makes suitable changes in the milk when he finds his fats too high or too low, according to their effect upon the child and not because he knows exactly what percentage exists in the milk used. Personally, I do not find that with the average healthy child it is any advantage, and, as ordinarily used, it is a very great disadvantage to add cereals to milk. I start with a very low percentage of fats and proteids and watching the effect upon the child's digestion carefully increase the percentages of both.

One principle has been lost sight of in most of these discussions, and that is the great advantage of a change of food in stopping the diseased process in the intestines. One baby has been fed on modifications of milk and does better for a time when cereals are added, and another who has had cereals in considerable amount improves when they are omitted and the baby is put on modified milk. It is largely a question of change and rest. While Keller's work is good I think the number of cases he has published is altogether too small to draw any sweeping deductions from them.

DR. WINTERS.—Like Dr. Chapin I have had experience in feeding infants with cereals but it was many years ago and it was with very indifferent results. I finally came to the conclusion that the fault was in not properly modifying the milk and that this could not be attained by the addition of cereals. By studying more carefully the home modifications and then getting down to the laboratory question I worked with accurate percentages and followed up the modifications with the utmost care. I do not believe that there is, has been, or will be, an infant that cannot be successfully fed from birth on a properly modified milk. I have not met with such a case in a large experience. I think the important point is that we should have a very definite relation between the fats and proteids rather than a definite percentage of fats and proteids. I doubt whether a baby can digest and thrive on a food containing but 1 per cent. of fat and find that you must have 2 per cent. of fat when you use .25 of proteids and a 3 per cent. fat with .50 proteids, etc. A baby will not digest, for any considerable time, 3 per cent. of fat with 1 per cent. of proteids; there must be some more definite relation between them. I believe these cereals tend in all cases to a disturbance of metabolism.

Perhaps the most serious point in this whole subject is that after the publication of this paper Dr. Chapin's words will almost certainly be used as an advertisement by some of the proprietors of infant foods.

DR. HOLT.—I would like to ask Dr. Winters if he has ever started an infant on 1 per cent. of fat and .33 of proteids.

DR. WINTERS.—I have tried it and they do not do well for any considerable period, say for a month.

DR. HOLT.—Well, I have often seen babies that have been treated with such proportions as 4 per cent. of fat and 1 per cent. proteids, where much vomiting and other serious symptoms of indigestion resulted.

DR. SAUNDERS.—Undoubtedly the cereals are very important in the sick diet and whether they should be introduced into the normal child's diet is now the question under discussion. In cases

of abnormal digestive power we have to use cereals in order to supplement the whey proteids. As to the use of certain proportions of fat in milk mixtures, undoubtedly the fat which is incorporated in the meshes of the coagulum is one of the natural elements in the coagulation, and in the whey mixtures I see no reason for preserving any ratio between the fat and proteids. I should put whey down as the basis of normal foods and the cereals as a basis of sick foods. We have always known that there must be some organic acid present in milk whey and I want to call attention to the fact that it has been recently determined that it is citric acid which is, of course, the important anti-scorbutic element in this food.

DR. CHAPIN.—In regard to percentage feeding I want to give Dr. Rotch full credit for bringing it before the profession, for the benefit it has secured to a large number of babies and for his insistence upon our accepting it. I recognize the value of percentage feeding and employ it. The only criticism I should offer is that it has, perhaps, been pushed too far, and that certain other elements in the question which are exceedingly important have been relegated to the background.

With reference to the caseinogen and the absorbable proteids there is a difference of opinion by various authorities, Koenig, Richmond and others, and the whole subject is *sub judice*.

As to dextrinizing gruels, one great advantage is that we can use a much stronger gruel when dextrinized than when used plain and the breaking up of the curd is not the sole point at issue. It does that, but it does more, in that it furnishes a quickly absorbable nutriment that saves your proteids.

I don't believe the infant food makers will use this paper much; they certainly will not if they comprehend it all. These foods may do temporary good and I am trying to place in the hands of the profession a method of scientifically preparing cow's milk, so that they will be independent of the food manufacturers.

Dr. Holt spoke of using very low amounts of proteids at first. Colostrum is very high in proteids and experience shows in studying animals that the higher the proteids the more rapidly do the animals increase in weight. Dr. Winters says that babies never do well with the proportion of fats to proteids of 3 to 1, but that is exactly the proportion found in mothers' milk.

A CONTRIBUTION TO THE PATHOLOGICAL ANATOMY OF SPORADIC CRETINISM.

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In the latter part of 1897, Mr. S., of Reading, Pa., consulted one of us (Packard) because of hepatic insufficiency. In obtaining the routine history of this case it was found that one of his children, aged six years, was idiotic and undeveloped. Further questioning confirmed the opinion at first formed, that this might be a case of cretinism. He was urged to bring his child to the city, where it could be properly placed upon thyroid treatment. Accordingly, the child was admitted to the Children's Hospital on December 7, 1897. Nothing in the family history had any bearing upon the case. Two other children, born respectively before and after this child, were perfectly healthy. Although he was not weighed at birth, the subject of the paper seemed to be of normal size and development. He was breast-fed until he was twenty months old. At four weeks of age, while in his mother's arms, he had some sort of an attack in which he seemed to lose all power in the body and had complete arrest of respiration. He was taken to the fresh air, and soon revived. From this time it was noticed that his tongue protruded constantly from his mouth, and after this attack until he was two years old he cried almost constantly, sleeping only at intervals of a half hour's duration. It was noted that he grew very slowly, but at no time was it observed that he had distinctly ceased to grow. He was weaned at the age of twenty months, and was then fed on water crackers, soft-boiled eggs, and milk until three years of age, when he was given ordinary children's diet. For his whole life he has been intensely constipated, his bowels never moving more than three times a week. From the age of three to five years he never had a bowel movement without the use of a soap-and-water enema. During the early part of the period last mentioned he had obstinate constipation for ten days, in spite of efforts applied both by the mouth and the rectum.

When admitted, although six years of age, he measured only 81

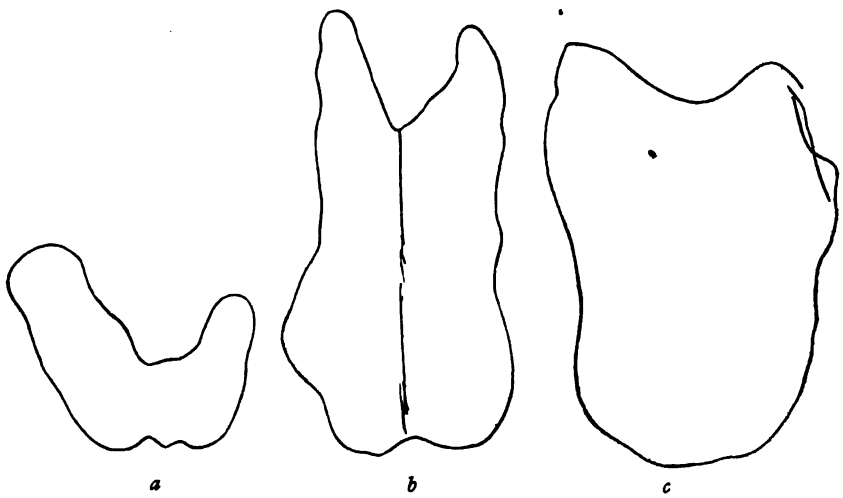
cm. He was unable to talk, and understood nothing but a few words, and those only when spoken with a certain tone of voice. He lay back among the pillows, perfectly apathetic, and not moving except for an occasional slight rolling of the head. The skin was of a peculiar yellowish-white color, and the mucous membranes were quite red. The features were heavy and doughy, the eyes half-closed with thick lids. The nares were widely opened, the alæ being decidedly thickened. The tongue was held constantly protruding from the mouth, and measured 4 cm. in width and 1½ cm. in thickness, completely filling the orifice of the lips, and being very doughy to the touch. He had a full set of temporary teeth, but they were stunted, wide apart, and most of them plainly decayed. The lips were very thick, the angles of the mouth rounded. The pupils were equal and natural, the eyelashes well developed. The scalp was almost completely devoid of hair, and had over it in places thick, oily, yellowish crusts. The anterior fontanel was widely open and pulsating. The ears appeared of a size proportionate to the rest of the body. The circumference of the head was 50 cm., the neck was very large and full, and at the base was surrounded with a thick collar of doughy consistence, measuring 32 cm. in circumference. The whole appearance of the face was distinctly porcine and devoid of expression. The tissues of the upper extremity were very thick and heavy, especially in the forearms, which felt as though they were the seat of solid edema. The hands were pudgy and short, and the skin of the hands curiously wrinkled. The trunk had a curious shape, owing to the huge development of the soft parts, and the small relative size of the hips. The chest was round, much expanded at the base, and the costosternal angle formed almost a straight line. Owing to the thickness of the neck the condition of the thyroid gland could not be made out, although it was not apparently enlarged. Careful examination of the lungs, heart, liver, and spleen showed no abnormality. The abdomen was very aldermanic, and there was a quite large umbilical hernia. The spine was straight except in the lumbar and lower dorsal regions, where there was distinct bowing. The genitalia were well developed, both testicles descended, and the prepuce was rather long. The lower extremities showed distinct bowing, with great rolls of tissue at the normal flexures. The feet were noted to have the same curious wrinkled appearance that was present in the hands. It was soon

found that while he could take solids fairly well it was impossible for him to swallow liquids. His weight on admission was twenty-seven pounds eleven ounces. Four days after admission he was placed on two grains of thyroid extract three times daily. This was continued until January 2d, at which time he had lost in weight three pounds and thirteen ounces. Even as early as nine days after admission decided improvement was noted, and he was found in a feeble way attempting to amuse the children in the ward by throwing things on the floor. By December 23d it was noticed that the tongue was less constantly protruded, that he frequently smiled and looked around the ward, and that he was able to take fluids much better than on admission. By December 28th, three weeks after admission, his skin was becoming wrinkled and the collar of fat was rapidly disappearing. On January 2d the dose of thyroid extract was diminished, owing to an apparent weak attack which he had had on that morning. One grain was given three times daily from January 2d until January 16th. On the latter date he weighed twenty-two pounds two ounces, a loss of five pounds nine ounces in the five weeks since his admission. During this period he steadily improved as regards his general appearance, while his mental condition had showed a manifest change. On January 12th he was found to measure 2½ cm. more in length than he did on admission, about one month before. On January 16th the dose of thyroid was decreased to one-half grain, and on the 19th it was reduced to one-quarter of a grain three times daily. On January 22d he weighed twenty-one pounds four ounces, the lowest weight recorded up to that time. Owing to his seeming rather weak the dose of thyroid gland was diminished to one-quarter of a grain daily from January 29th to February 10th. On the 20th of the latter month his length was 85 cm., a growth of 4 cm. since his admission. His weight was twenty-one pounds eight ounces, or six pounds and three ounces less than on admission. With his improvement in other directions his bowels also showed a greater tendency to move naturally. He was kept on the thyroid extract in doses of one-quarter of a grain two or three times daily until April 14th, when he was taken back to his home. When he left the hospital, in the middle of April, he had ceased losing weight, was bright and active, took an interest in his surroundings, recognized his favorite nurses, cried when he considered himself slighted, and in every way acted quite naturally.

He showed himself to be of a very affectionate disposition, and recognized with pleasure the return of his parents, whom he had not seen for some months. While he was at his home he seemed to gain even more as regards his mental power. He there played with the other children, learned to feed himself, and gained about two pounds in weight. He was brought back to the hospital on May 6th, and was found to weigh twenty-five pounds. On his return to the hospital he seemed to recognize the nurse who had taken care of him before, and, as nearly as could be told, remembered his surroundings. On his admission he was again put on the extract of thyroid in doses of one-quarter of a grain three times daily. His weight on readmission was twenty-four pounds. The thyroid extract was continued, but he was sent to the country branch, where he gained in weight, apparently from his more healthy surroundings in the fresh air. He remained in the hospital on this occasion until July 26th, when he was discharged, looking very different than on his first admission. He was readmitted to the hospital on October 20, 1898. While at home he had been doing very well, treatment being continued. He was learning to talk, was constantly creeping about, and trying to stand with the help of a chair, and certainly understood much that was said to him. During his stay at home he gained over six pounds in weight. His length had increased between the date of his first admission, December 7, 1897, and the date of his readmission, October 20, 1898, to the extent of 9 cm. Reexamination on his last admission showed that the general appearance had decidedly improved; that the hair was well grown; that the anterior fontanel was almost entirely closed, and that the tongue was kept constantly within the mouth. The abdomen was very much less distended, and the relative sizes of the chest, abdomen, and pelvis were much nearer normal. He stood quite well when given slight support, he laughed quite naturally and merrily, and in place of the feeble whine or grunt with which he expressed displeasure on his first admission the cry of anger or of grief was quite natural. He was again put on thyroid extract in doses of from one-quarter to one-half grain three times daily, and this was followed by some loss of weight. Except for this, however, no material change occurred, until on November 5th he developed elevation of temperature, dry cough, abdominal distention, and coated tongue. The temperature gradually rose until it reached a range of from

104° to 105.5°, with occasional elevations even beyond that point. From the very onset of this acute attack he rapidly emaciated, became intensely adynamic, and died on November 12th, one week after the onset of his acute illness. During the whole course of this attack it seemed as though the resisting power was almost *nil,* and from the very onset it was evident that his illness was almost certain to terminate fatally.

AUTOPSY was performed three hours after death. Rigor mortis was present. There were dark purple ecchymoses, mainly over the left shoulder and upper arm, over both knees, and over



Courtesy of American Journal of Medical Sciences.

FIG. 1.—Actual length and breadth of thyroid and thymus glands. (*a*) Thyroid body. (*b*) Cervical portion of thymus. (*c*) Mediastinal portion of thymus.

the left tibia. Subcutaneous fat was well marked. The lungs were normal. Directly below the thyroid cartilage there was a body resembling the thyroid gland in external appearance, with a narrow constriction in the centre and two wings which nearly encircled the trachea, the right lobe being the larger. From the lower part of the body a vein ran directly downward into the left innominate. Below this evident thyroid body there were two tips of glandular substance in the position of the lower part of the normally placed thyroid gland. These bodies extended into the neck 1½ cm., and were the extension of a large mass of glandular

tissue situated in the anterior mediastinum. The cervical part was composed of two lobes held by connective tissue. They showed on microscopical examination the typical structure of thymus gland. In the anterior mediastinum was another large mass, which also was composed of normal thymus tissue. The bronchial glands were not enlarged. The spleen was firm, pale red, and rather dry. The liver reached to the costal margin and had the appearance of being fatty. The kidneys were lobulated, the cortex pale, and with marked stellate injection of the veins. The intestines showed on the peritoneal surface areas of congestion. In their lumen there was a small amount of yellowish-green fecal matter. From the jejunum down Peyer's patches were elevated and red, while some were distinctly ulcerated. Throughout the ileum there was widespread and intense folliculitis. The esophagus showed injection of the mucous surface which felt like very fine sand-paper. The stomach was normal, as were also the pancreas and the suprarenals. In the superior longitudinal sinus of the brain there was a yellow thrombus. Nothing important was noted in the brain, except for the fact that the hypophysis measured 13x7x5 mm., which was considered abnormally large for a child of this size and age.

Microscopical Examination. Thyroid Gland.—The alveoli are distinctly marked off by bands of white fibrous tissue, which is in greatest amount around the blood vessels. The epithelial cells are abundant, showing considerable variation in size. The acini are small, many contain no colloid substance whatever, and in those in which the colloid is present it is in very small masses, the total amount in the section being scarcely more than that in two or three acini of a normal thyroid. Equally striking with the deficiency in colloid is the presence in the walls of the larger blood vessels, especially the veins around the gland, of a decided calcareous change. This involves especially the media, with the formation of cavities surrounded by the calcareous deposit; in some places the process has extended to the adventitia and the intima, but in none of the sections was there visible a rupture of any of these "atheromatous abscesses" into the lumen of the blood vessels. (Figs. II. and III.)

Thymus.—Both the upper and lower bodies of the thymus were normal, the cells staining well.

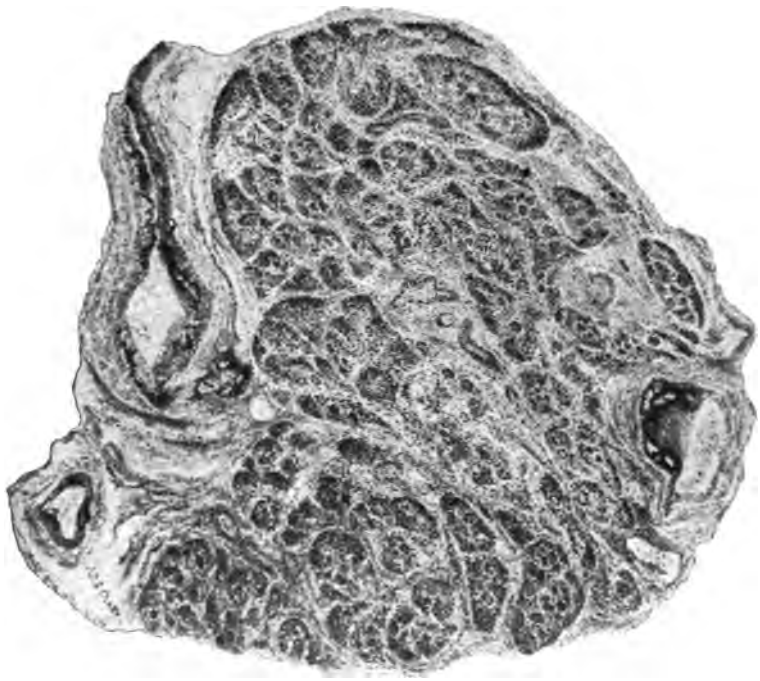
The changes in the other organs were such as could be attrib-

uted not to the cretinism, but to the febrile infection, which resulted fatally.

Hypophysis.—The acini are distinct and the glandular epithelium is abundant; the fibrous layer is scanty; the posterior lobe resembles the anterior.

Aorta.—Normal; no sign of calcareous change.

Skin from Abdominal Wall.—The horny layer is very thin;



Courtesy of American Journal of Medical Sciences.

FIG. II.—Thyroid gland, showing atrophy and increase of fibrous tissue; calcareous deposits in walls of large blood vessels.

the rete mucosum is normal in appearance. In the papillæ of the cutis vera there is a widespread infiltration of lymphoid cells, which in the corium show a tendency to form aggregations. The nuclei of the corium are numerous and stain well. The fibres of connective tissue of the corium are all very coarse, those near the papillæ being slightly finer. The elastic fibres are few in number. In the deeper layers of the corium there is decided infiltration of lymphoid cells around the ducts of the sweat glands.

Liver.—There is a universal fatty degeneration of the parenchyma with large areas in which the outlines of the cells are indistinct and the nuclei are imperfect. Distinct areas of focal necrosis are not made out, which would point to another than a typhoid infection.

Spleen.—All the nuclei stain poorly, and the cells are separated by a granular material.

Kidneys.—There is a marked degeneration of the parenchyma throughout all the tubules.

Courtesy of American Journal of Medical Sciences.

FIG. III.—Thyroid gland from a normally developed child of nine years.

Heart Muscle.—There is a very slight cloudy swelling.

Suprarenals.—The nuclei do not stain well. The layer of pigmented cells is not distinct.

Two or three points in this case seem to us to be worthy of more than passing mention. These we consider to be the rapid improvement under the use of the specific drug, thyroid extract; the extremely rapid malignant course of his infection, typhoid fever; the presence of enlargement of the pituitary body; the great increase in the size of the thymus gland; the presence of the

thyroid gland, and the extensive disease of this, with calcification of the walls of the arteries going to it. In addition to this the intensity of the lesions produced by the typhoid toxin, and especially the necrobiotic changes occurring in the liver, are worthy of mention, because of their possible bearing upon the question of the resisting power of cretins treated by thyroid extract.

The rapid improvement in the mental and physical condition under treatment with thyroid gland is so ordinary an occurrence as to require no extensive consideration. We can find in the literature but few facts bearing upon the relative resisting power to infections possessed by the cretin as contrasted with the normal human being. In the *Lancet* of December 10, 1898, Byram Bramwell particularly remarks upon the fact that a female cretin, aged thirty-six years, "had never had any of the diseases peculiar to childhood, though she had been exposed to some of them. She had slept, for example, in a room with one of her sisters who was suffering from scarlet fever during the whole course of the disease, but did not take it." Among the reports that we have read there is no other evidence of any peculiar immunity presented by the cretin. One case mentioned by Osler,[1] occurring in the Indiana School for Feeble-minded Children, died from acute tuberculosis. This case had been under treatment with thyroid extract from September, 1895, until its illness in March, 1896. It is difficult to estimate to what extent and in what manner the resisting power of the cretin to disease in general is affected by diminishing the cretinoid condition through the administration of thyroid gland. In our case it seemed as though the resisting power was very slight, and from the very onset the child rapidly lost flesh and strength, became intensely adynamic, and died at the end of the first week from asthenia—a termination of typhoid seldom seen in childhood.

Nicholson[2] reports a case treated with thyroid extract from October, 1894, to July, 1895, with marked improvement. In the middle of the latter month the child "contracted measles of a most malignant type, and died on July 16th, after three days' illness." Nicholson states that a sister died of the same disease at the same time, and that apparently the treatment of the condition of cretinism did not render the child more susceptible or less resisting.

1. American Journal of the Medical Sciences, October, 1897.
2. ARCHIVES OF PEDIATRICS, June, 1900.

We have been unable to find a sufficient number of cases thoroughly reported from which to draw conclusions as to the resisting power of cretins treated or untreated. Bourneville and Bricon[1] mentioned one cretin who passed successfully through attacks of erysipelas, and quoted a case of Charpentier's dying from erysipelas, and one of Ball's dying from "albuminuria with typhoid symptoms."

So far as we can find, the first mention of enlargement of the hypophysis occurring in a cretin was reported by Boyce and Beadles.[2] In this case the cretin was nine years old, and growth had been arrested at the end of the first year. No trace of the thyroid was found, but the pituitary body was enlarged, so that it measured 1 cm. by 6.5 mm. (the measurement of the cut accompanying their article). In an autopsy upon a cretin well-known among Philadelphia men, who for years was a patient in the neurological ward of the Philadelphia Hospital, the autopsy, at which one of us (Packard) had the opportunity of being present, showed that while the thyroid gland was absent the pituitary body was not enlarged. In almost all of the fatal cases of sporadic cretinism the thyroid gland has been shown to be entirely absent. In the case of Bartine, briefly mentioned above, a careful search was made for this organ, and its complete absence was thoroughly demonstrated.

In 44 cases of marked sporadic cretinism tabulated by Byram Bramwell[3] 14 were fatal. In 10 of these the condition of the thyroid gland was noted. In all but 1 of these 10 there was no trace of thyroid gland remaining. Among Osler's cases, collected in 1897,[4] he narrates the case of a cretin, aged fourteen years, dying at the Indiana School for Feeble-minded Children. This child had been treated with thyroid extract with marked improvement, but died of acute tuberculosis about a year after treatment was instituted. The thyroid gland weighed four grams, as contrasted with the normal weight of fifteen to twenty grams. Careful study of this thyroid gland, made by Dr. Barker, agrees in some respects very closely with the examination of the thyroid

1. Archives de Neurologie, September, 1886.
2. Journal of Pathology and Bacteriology, 1893.
3. Atlas of Clin. Med , Vol. I., p. 17.
4. Transactions of the Congress of American Physicians and Surgeons, Vol. IV., p. 169.

gland from our case. The enlargement of the pituitary body in the case which we report has not been very frequently noted. In addition to the case of Boyce and Beadles, mentioned above, where this gland was enlarged, we find one mentioned by Comte, quoted from Burckhardt,[5] wherein the sella turcica was said to be very large, and the hypophysis hard, reddish, and of the size of a bean, its weight when fresh being 0.36 gram. Microscopically it was found that it contained a large amount of colloid material both in the interior of the gland and in the canal with small masses of colloid material within the acini. It also showed very many vessels filled with blood. In regard to the relative weight of this gland in his case, he compares it to two others from patients aged five and six years (his case being three years), which weighed 0.180 and 0.315 respectively, as contrasted with the weight of 0.360 in his case. Comte mentions the examination of the hypophysis made by himself in the case of a cretin dying two days after birth, in which the hypophysis weighed 0.123 gram, and showed that the posterior or the nervous portion was composed, not of the ordinary nervous tissue of other hypophyses, but of the same form of tissue as composed the anterior lobe. In his case, also, the hypophysis was very vascular without excess of colloid material. The thyroid gland was present and weighed in the fresh state twenty-two grams; microscopically it appeared to be very vascular and contained but a very small quantity of colloid substance. Particular mention is made of these few facts in regard to the hypophysis in cretinism since the observations of Rogowitsch, which seemed to show a compensatory action of the pituitary body in cases of experimental removal of the thyroid gland in animals.

A point which seems to us of more interest than any in connection with this case is the calcareous infiltration of the walls of the blood vessels of the thyroid body. While we have been unable to make as thorough an examination into the literature of the subject as we could have wished, we have failed to come across any mention of such a change observed by others. Its significance cannot be definitely asserted, but the observation is of some interest because of the supposed relation between various diseases connected with the thyroid gland and the drinking water. We have written to many of the physicians in the town from which this boy came, but have but few replies in answer to our inquiries

5. Ziegler's Beiträge, 1898, Bd. XXIII., p. 104.

regarding the frequency of goitre, myxedema, and cretinism in that locality. One surgeon with a large practice has written to us that he constantly has 2 or 3 cases of goitre under his care. His answer has been so recently received that we have not been able to obtain, more exact particulars from him. Although an unscientific opinion, it may be mentioned that the father of this boy states that he knows 2 cases similar to his son's among people living in his immediate neighborhood. He has endeavored to persuade them to have their child placed under proper treatment, but without avail. This statement, of course, should not have much weight, but the family likeness of cretinism is so close and the appearance is so characteristic that a lay diagnosis of one painfully familiar with the picture might be received with consideration.

DISCUSSION.

DR. COTTON.—I was very much interested in Dr. Packard's report although I did not quite understand his statement of the relationship of the rising temperature to the administration of the thyroid extract. Two weeks ago I presented to the Chicago Medical Society 2 cases of cretinism (one seen in private practice and the other at the Presbyterian Hospital), emphasizing the apparent effect of thyroid treatment on the temperature in its relation to the dose administerd. In each case the child improved, losing in weight and gaining in intelligence, but upon the gradual increase of the dose there was a gradual (occasionally a sudden) rise of temperature. In addition, and this, it seems to me, is in line with the idea of the writer, concerning the diminished resistance that these creatures exhibit as they lose their distinct cretinism, these cases both developed influenza. One case first seen last August, was put upon thyroid extract, but was taken home after less than five weeks' treatment with the presumption that the treatment would there be continued. Subsequently I learned that the thyroid had been discontinued because of a severe bronchitis which the parents thought might have some relation to the medicine administered. In February last the patient was returned to the hospital. The treatment was continued with gradually increasing doses until five grains were taken three times a day. The little fellow became quite ill with an attack of influenza which subsided when the treatment was stopped. Treatment was resumed, and again the temperature rose with the increased dosage and this time a bronchitis resulted. The temperature chart showed an apparent relationship between the fever and the increase in dosage.

The case in private practice was similar in this, that the parents themselves continued the treatment for several weeks. Later, my assistant called and found the child in quite a precarious condition. He made the diagnosis of influenza, discontinued the treatment and after a week's time, when I saw the case the temperature was normal. I would like to know if others have had this sort of experience. Is not the conclusion plain that in the administration of thyroid for cretinism there should be a most careful supervision?

DR. KOPLIK.—Concerning the resistance of cretins, I have found that there are several cases in literature that have died very rapidly after contracting pneumonia or diphtheria. I have had cretins under treatment with intercurrent pneumonia and can say that they do not all die. They show much less resistance to processes involving the lungs than do other children. In other words they seem more sick than normal children attacked with the same disease.

DR. NORTHRUP.—I have also had a case of pneumonia in a typical cretin who got through the acute disease satisfactorily.

DR. CHRISTOPHER.—We had a cretin in the Foundling Asylum some fifteen years ago who was noted as the only child in the institution who did not have diarrhea; every baby there had it except this one who was there altogether about a year. This was previous to the time when I, at least, knew anything about the treatment of cretinism so the patient was without any definite treatment.

I had a cretin under treatment, who, two weeks after it was begun, developed an acute pulmonary tuberculosis that carried him off very rapidly. I do not recall any other cases of sickness in cretins that have been under my care.

The point raised by Dr. Cotton as to the elevation of temperature leads me to say that I think there is a marked tendency for the thyroid extract to produce an elevation of temperature and, furthermore, that it is a very common thing for these children to have disturbed sleep and I have been unable often to run up the doses necessary to secure good results on account of these disturbances. The preparation I use is the Armour dessicated thyroid and I find that one or possibly two grains twice a day is about all that the cretins under my care will stand. I have also noticed that my cretins do not get well. They make a very rapid improvement for a time but it does not continue to increase in the same ratio. I have one little girl under my care now at seven years of age that I have treated with the thyroid since she was seven months old and she is far from being a normal child. She is deficient mentally, not, of course, as she was, though she had improved physically. I should be glad to hear whether the other

gentlemen have had their cretins go continuously and rapidly forward to become something like normal individuals.

DR. WEST.—I began the treatment of a cretin about fifteen months ago and after she had been under treatment about six weeks she developed a severe bronchitis and it was only by the most careful nursing that her mother gave her that she got well at all. For three months afterwards she did not get back anywhere near what she was before the advent of the bronchitis and I really think that disease has had an effect upon her ever since because I have not gotten the results with thyroid in this case that I have usually attained.

The cretin whose case I reported a number of years ago seems to have very strong resistant powers. She had a very severe attack of measles last winter and survived while her normal brother died from the effects of a similar attack. I have known also that she stands a great deal of exposure for I saw her recently sitting out near the barn in a cold drizzling rain which seems not to have affected her.

DR. BOOKER.—Are you still giving the thyroid to that first case?

DR. WEST.—She gets it irregularly.

DR. BOOKER.—How is she now?

DR. WEST.—Of course very much better than she was, but she grows very little. Her mind improves more than her body, although it is not what it should be.

DR. NORTHRUP.—I would like to testify concerning the point that Dr. Christopher made that these patients make a very rapid, sudden improvement which continues for five or six weeks and then there follows a long, doubtful period during which we cannot tell whether we are accomplishing anything. Of 2 cases I took into the hospital, one developed pneumonia and the other became very flabby and we soon learned that they do not thrive in the hospital. We have 5 or 6 cases coming to the hospital clinic, however, and in the course of a year they make such changes that I fail to recognize them when I see them. Two of them have done very well, while 2 have not done anything at all and I suspect the treatment has not been regularly conducted.

DR. KOPLIK.—I think success with these cases depends on how soon the treatment is instituted. If we get the cases early they develop well and look almost like normal children. If the cases come under treatment late (the second year) the result is unsatisfactory. To give an example, one boy now six years old, can only sing his A, B, C's, although he made very rapid improvement at first. He came under observation at the nineteenth month. Improvement soon stopped. Thomson, of Edinburgh,

has reported some cases that improved sufficiently to be sent to school with other children. If the cases come under our care as in some of Thomson's cases, late, the thyroid treatment causes an additional complication in the shape of softening of the shafts of the long bones.

DR. FREEMAN.—I have had a cretin under treatment for the past year that gives a history very much like those related; that is to say, after a few months of treatment the child developed a bronchopneumonia and became very sick, but recovered. At the present time it has none of the appearances of the cretin but at the same time it is backward mentally. It is two years old and does not talk at all, but since that first illness has remained in a healthy condition.

DR. CHRISTOPHER.—There is a cretin in Chicago, a colored boy who was about twenty-one when I first met him, and he has a sister who is also a cretin. The boy refused treatment, but the sister took it and made some slight improvement. She has continued treatment more or less irregularly for four years but her condition relapses whenever the treatment is stopped. I do not know whether there are many cases of cretinism in the colored race and I would like to put these two on record.

There is a peculiar feature about some of these cretins which may be a symptom of the disease and that is the growth of hair low down on the forehead; that phenomenon always makes me think of cretinism.

DR. NORTHUP.—I would like to ask what is the relation of consanguinity to cretinism.

DR. PACKARD.—I think Dr. Osler, in his tabulated report of cases, said that very few cases occurred in which there was any relationship between the parents.

DR. ADAMS.—Are we to interpret this discussion as a reaction against the treatment by thyroid extract?

DR. KOPLIK.—I think not. We may not cure these cases entirely, but we make them useful members of society.

DR. PACKARD.—In going over the literature of the subject of cretinism and its pathological anatomy, it was noteworthy that very few autopsies had been held. We were also struck with the fact that while a few years ago the journals contained many reports of cases of cretinism these appeared but rarely of late, and that no reports have appeared showing the subsequent course of those reported some years ago. The question of improvement with prolonged treatment can, so far as the literature shows, be no more definitely determined now than it was soon after the method of treatment was discovered. The rise of temperature

after the administration of thyroid gland, which has been spoken of in the discussion, was present in our case. Usually, however, the transient elevations of temperature we found to be due to some infection of the upper respiratory tract. These frequent slight infections of the upper respiratory tract and the intensity of the infection by the typhoid bacillus was the cause of our making some attempt to find some evidence in the literature as to the cause of death in cretins untreated by thyroid extract, and also the possible diminution of resisting power brought about by the administration of thyroid gland. In regard to the question of the possibility of bringing about improvement in these cases, I think with one of the speakers that it depends almost entirely upon the age at which the patient comes under treatment. The case that I briefly spoke of as being an inmate of the Philadelphia Hospital for a very long time was forty years old when thyroid treatment was instituted. The dose in this case was increased up to the physiological limit with absolutely no effect upon the original condition.

INDEX

TO THE

Transactions

OF THE

American Pediatric Society.

VOLS. I.–XIV., INCLUSIVE.

COMMITTEE ON INDEXING
THE TRANSACTIONS.

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L. Emmett Holt, M.D.,
Rowland G. Freeman, M.D.

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1903.

AMERICAN PEDIATRIC SOCIETY.

HISTORICAL TABLE.

YEARS.	PRESIDENTS.	PLACES OF MEETING.	PRESIDENTIAL ADDRESSES.
1888		Washington.	Organization of American Pediatric Society.
1889	A. Jacobi.	Washington and Baltimore.	The Relation of Pediatrics to General Medicine. . I.–6.
1890	J. Lewis Smith.	New York.	The American Pediatric Society—Its Present and Its Future. . . . . II.–6.
1891	T. M. Rotch.	Washington.	Iconoclasm and Original Thought in the Study of Pediatrics. . . . III.–6.
1892	William Osler.	Boston.	Specialism. IV.–6
1893	A. D. Blackader.	West Point.	A Review of the Work of the American Pediatric Society. V.–8.
1894	John M. Keating. Died in Office.	Washington.	
1895	F. Forchheimer.	Hot Springs, Va.	A Pediatric Poem. . VII.–7.
1896	Joseph O'Dwyer.	Montreal.	The Evolution of Intubation. VIII.–9.
1897	Samuel S. Adams.	Washington.	The Evolution of Pediatric Literature in the United States. IX.–5.
1898	L. Emmett Holt.	Cincinnati.	The Scope and Limitations of Hospitals for Infants. X.–147.
1899	William P. Northrup.	Deer Park, Md.	Methods of Instruction in Pediatrics. . . . . XI.–7.
1900	Henry Koplik.	Washington.	The Management of the Gastro-Intestinal Derangements of Infancy. . . XII.–7.
1901	William D. Booker.	Niagara Falls.	The Early History of the Summer Diarrhea of Infants. XIII.–7.
1902	W. S. Christopher.	Boston.	Development the Keynote of Pediatrics. . . XIV.–9.

Index by Authors.

ADAMS, SAMUEL S.

Papers and Cases Reported:

Aphasia, amnesic, scarlatina, pneumonia, VII-136; brain, abscess of, VIII-126; carbolic acid, rapid absorption of, by eczematous skin, VI-134; diphtheria antitoxin in private practice, VIII-21; diphtheria antitoxin in District of Columbia, VIII-46; diphtheria antitoxin in laryngeal cases, IX-32; empyema, irrigation in, by submersion, X-80, endocarditis, septic, XIV-160; erysipelas, V-199; insanity, temporary, following typhoid fever, VIII-177; laryngismus stridulus, death from, in incipient rickets, IV-190; malaria, XIII-236; pediatric literature, the evolution of, in the United States, IX-5; pharyngo-esophageal stenosis, VIII-166; pulmonary tuberculosis, temperature in, XIII-184; rheumatism and chorea, IV-231; sarcoma of the cerebellum, XI-69; vapo-cresolene, poisoning by, XII-174.

Discussions:

Chorea and rheumatism, IV-231; circulatory failure in acute infectious disease, XIV-193; diarrhea, summer, XIV-41; diphtheria antitoxin, VIII-68; empyema, X-84; endocarditis, XIV-173; enteric fever, XII-107; erysipelas, V-204; feeding, artificial, I-84; german measles, X-126; grippe, IV-34; heart, malformation of, XII-146, 147; insanity, temporary, following typhoid fever, VIII-185; insolation, VIII-208; intestinal obstruction, XII-70; intubation, XIV-136; intussusception, XIV-30, 31; laryngismus stridulus, IV-195, 196, 197, 199; malaria, XIII-236; masturbation, VIII-188; milk, separation of bacteria from, XI-39; nephritis, X-145; paralysis, epidemic, XII-162; pediatrics, method of instruction in, XI-21; peritonitis, tuberculous, XIV-105; playrooms on city roofs, XIII-223, 224; pneumonia, III-57; XIV-209; renal complications of enteric disease, XI-58; rickets, VI-173; retro-esophageal abscess, IX-179; scarlatina, VII-108; scurvy, X-29; XI-181; stomatitis, III-253; summer diarrhea, XIII-48, 57; XIV-41; tuberculosis, XIII-191, 192; typhoid fever, IV-143; urine, incontinence of, V-167; weight, increase of, in infants artificially fed, XI-118.

ACKER, GEORGE, N.

Papers and Cases Reported:

A cardiac anomaly, VII-155; arthritis, multiple, XIII-238; gangrene of the lungs complicating typhoid fever, VIII-141; hemoptysis, a fatal case of, X-89, 137; heart disease following scarlet fever, VIII-163; lympho-sarcoma of the spleen, VII-73; malarial coma, XII-163; meningitis, tuberculous, IX-75; mirror

writers, VI-79; umbilical fistulæ due to tuberculous peritonitis, XIV-110; porencephalus, XI-131; rickets in negroes, VI-137; sarcoma of thymus and bronchial glands, VIII-214.

Discussions:

Circulatory failure in acute infectious disease, XIV-191, 193; meningitis, tuberculous, IX-82, 83; peritonitis, tuberculous, XIV-105; rickets, VI-176; scarlatina, VII-108.

BAINES, ALLEN.

Two fatal cases of lead poisining, XII-122; arteriosclerosis, a case of, XIII-58.

BARKER, LEWELLYS F.

A case of acute diffuse scleroderma in a child of two and a half months, V-142.

BLACKADER, A. D.

Papers and Cases Reported:

American Pediatric Society, a review of the work of, V-8; ataxia, I-191; enteric fever, XII-98; liver, marked enlargement of, with symptoms of typhoid fever, VIII-239; scarlatina, eruptions simulating, VII-76.

Discussions:

Circulatory failure, XIV-191; diarrheal disease, II-128; enteric fever, XII-107, 116; feeding, artificial, XI-119; grippe, IV-36; VII-144, 145, 146; gruels, XI-224, 225; heart, malformation of, XII-147; influenza, II-207; intussusception, XIV-32; laryngismus stridulus, IV-195; liver, lesions of, XI-252; lumbar puncture, VIII-110; ozone, IV-21; pancreatic digestion of casein, XII-43; pediatrics, methods of instruction in, XI-19; peroxid of hydrogen, IV-213; rheumatism, XIV-70; trachea, perforation into, XII-96; typhoid fever, IV-138; urticaria of the mucous membranes, XI-109; weight, increase of, in artificially-fed children, XI-119.

BOOKER, WILLIAM D.

Papers and Cases Reported:

Diphtheria, etiology of pseudo-membranous inflammation—general distribution of the diphtheria bacilli, V-24; diphtheria, its relation to pseudo-diphtheritic angina (scarlatinal), IV-45; dysentery, ulcerative catarrhal, III-192; hernia, diaphragmatic, IX-100; summer diarrhea, bacteria of, I-198; summer diarrhea, early history of, XIII-7.

Discussions:

Diarrheal diseases, I-311; II-122; diphtheria antitoxin, VII-52; VIII-68; diphtheria, nasal feeding in, VIII-78; empyema, II-59; feeding, artificial, I-83; gastrointestinal disturbances, mechanical treatment of, I-246; grippe, IV-36; hernia, diaphragmatic, IX-111; inanition fever, VII-65; masturbation, VIII-189; purpura, II-164; respiration, artificial, III-132; rheumatism and chorea, IV-241; scurvy, X-25, 26, 31.

BOWEN, J. T.

Discussion:

Alopecia areata, IV-42.

BROWN, DILLON.

Papers and Cases Reported:

O'Dwyer's tubes, the construction of, with report of 350 cases of intubation, II-182; Pott's disease, early diagnosis of, VI-89; respiration, noisy, I-43.

Discussions:

Croup, membranous, I-69; diphtheria antitoxin, IX-38; diphtheria following tonsillotomy, VI-72; meningitis, tuberculous, IX-83.

BUCKINGHAM, E. M.

Paper:

A clinical study of cases of angina resembling diphtheria, the bacilli being reported absent, VII-186.

Discussions:

German measles, X-125; grippe, VII-145; intubation in diphtheria, XIV-135; laryngeal stenosis, XIII-276; malaria, XIII-236; milk proteids, XIV-154; peroxid of hydrogen, IV-214; scurvy, X-26; summer diarrhea, XIII-46.

CAILLÉ, AUGUSTUS.

Papers and Cases Reported:

Circulatory failure in acute infectious diseases, XIV-190; croup, membranous, I-67; chart and record for clinical diagnosis, XI-142; diphtheria antitoxin, VII-38; diphtheria following tonsillotomy, VI-66; diphtheria, general prophylaxis in, II-136; diphtheria, personal prophylaxis in, I-108; heart, transposition of great vessels of, VIII-168; janiceps asymmetros, I-70; laryngeal stenosis, VIII-232; lumbar puncture, VIII-79; nephritis, chronic parenchymatous, XIV-120; nystagmus associated with choreic movements of the head, I-237; ozone, value of nascent, IV-14; prolapsus-recti from stone in bladder, I-157; pyothorax, X-54; sarcoma of suprarenal capsule, VII-75; scurvy, X-24; thymus gland, sudden death due to enlarged, XIV-194; trachea, perforation of bronchial lymph node into, XII-94; tuberculous peritonitis, operative treatment of, XII-17.

Discussions:

Anemia, V-141; appendicitis, XIV-119; broncho-stenosis, syphilitic, IV-187; cardiac lesions, VIII-170; circulatory failure in acute infectious diseases, XIV-191; diphtheria, V-34; diphtheria antitoxin, VIII-66, 70; X-117; XIV-133; diphtheria following tonsillotomy, VI-74; diphtheria, tracheal and bronchial casts in, IV-92; empyema, I-26; X-84; enanthem, XIV-74; feeding, artificial, I-84; gastrointestinal disturbance in infants, mechanical treatment of, I-249; gastroenteric tract, nomenclature of diseases of, VI-58, 59, 61; gastric neurosis, V-194; german measles, X-127; grippe, IV-33; VII-145; hydrorrhachis, etc., IV-107; hyperpyrexia, VII-149; incubators, V-46; intestinal obstruction, XII-70; intubation in diphtheria, XIV-133; intussusception X-107; XIV-27; laryngismus stridulus, IV-195, 196; larynx, stenosis of, VIII-233; lumbar puncture, VIII-111; malaria, I-185;

meningitis, II-85; meningitis, cerebro-spinal, VIII-201, 202; meningitis, tuberculous, II-85; milk, preservation of, V-43; filtration of, VI-39; nephritis, X-144; XII-141; O'Dwyer's tubes, II-197; otitis media, VII-135; ozone, value of nascent, IV-19, 20, 21, 22; pancreatic digestion of casein, XII-43, 44; papillomata of larynx, VIII-162; pericarditis, VIII-170; peritonitis, tuberculous, XII-27; XIV-106; peroxid of hydrogen, IV-212, 213; peritonitis, I-127; pertussis, V-125; pyelitis, VI-133; respiration, noisy, I-55; sarcoma of suprarenal capsule, VII-76; scarlatina, VII-106; scurvy, X-25, 30; thymus gland, death due to enlarged, XIV-194; trachea, perforation of bronchial lymph nodes into, XII-94, 95.

CARR, WALTER LESTER.

Papers and Cases Reported:

Measles, X-194; pneumonia, a case probably due to streptococcus infection, XIV-205; pulmonary gangrene, XIII-256; pyopneumothorax, VII-69; rachitis, some manifestations of, without severe bone lesions, II-33; sarcoma of the skin of the back, V-204; tetanus neonatorum, VII-153.

Discussions:

Empyema, I-27; II-32; emphysema, I-66; hemorrhages of the newly-born, XIII-281; intestinal obstruction, XII-71 lung, re-expansion of collapsed, I-39; malaria, I-183, 186; medication, II-203; nephritis, III-232; XII-139; pneumonia probably due to streptococcus infection, XIV-209; respiration, noisy, I-55; rheumatism, XIV-69; rickets, VI-175; sarcoma of the cutis, IX-156.

CHAPIN, HENRY D.

Papers and Cases Reported:

Brain, multiple tumor of, VIII-236; cereals in infant feeding, XIV-229; diarrhea, summer, local variations in the mortality of, XIV-34; gruels as diluents of cow's milk, XI-219; hyperpyrexia, VII-146; intestinal obstruction, symptoms of, VI-63; meningitis, tuberculous, X-219; milk, preservation of, V-35; milk, the proteids of, XIV-149; paralysis, epidemic, XII-158; pertussis, complications of, V-126.

Discussions:

Alcohol, II-155, 156; bronchial nodes, enlarged, XI-68; cereals, XIV-244; cerebral abscess, XII-62; diphtheria antitoxin, VII-54; empyema, IX-89; XIII-218; gruels, XI-224, 229; hemorrhages of the newly-born, XIII-281; hyperpyrexia, VII-150; incubator baby, feeding of, XIII-132; intubation in diphtheria, XIV-134, 136; intussusception, XIV-29; lumbar puncture, XI-239; milk, preservation of, V-41, 43; milk proteids, XIV-152, 153, 159; paralysis, epidemic, XII-162; pediatrics, method of instruction in, XI-18, 20; peritonitis, tuberculous, XIV-106; renal complications of acute enteric disease, XI-59; retro-esophageal abscess, IX-177; rheumatism, XIV-68; scarlatina, VII-106; summer diarrhea, XIII-47; XIV-41; viscera, transposition of, XI-35; weight, increase of, in artificially-fed children, X-119.

CHRISTOPHER, W. S.

Papers:

Development the key-note of pediatrics, XIV-9; nutritional element in the causation of neuroses, VI-149; typhoid fever in infancy, IV-134.

Discussions:

Anemia with enlarged spleen, XIV-89; antipyretic drugs, V-75; autointoxication with neutral magnesium phosphates in the urine, XIV-189; cerebro-spinal meningitis, VIII-201; chondrodystrophy fetalis, XIV-57, 58; enanthem, XIV-75; filtration of milk, VI-40, 41; gastric neurosis, V-192, 195; inanition fever in the newly-born, VII-64; intestinal obstruction, XII-70; intussusception, XIV-32; nomenclature of diseases of the gastro-enteric tract, VI-61; proctitis, V-63; scurvy, X-24, 25; temporary insanity following typhoid, VIII-185; tonsillar infection, XIV-100; urine of healthy children, X-99.

CHURCHILL, FRANK SPOONER.

Papers and Cases Reported:

Albuminuria, cyclical, XIII-93; sarcoma of the kidney in an infant, X-178; the urine of healthy children, X-91.

Discussions:

Albuminuria, cyclical, XIII-110, 111; anemia, pernicious, XIII-90; acute nephritis following influenza, XII-140; the urine of healthy children, X-99, 100.

CONNER, PHINEAS S.

Discussion:

Intussusception, X-106.

COPLIN, W. M. F.

Paper:

Congenital cystic kidneys, XI-127.

COTTON, A. C.

Papers and Cases Reported:

A case of anuria, XI-137; a case of heart lesions, XI-196; diapers, the effect of tight, XIV-174; general subcutaneous emphysema, XII-128; congenital cardiac malformation with endocarditis and anuria, XII-142; idiocy amaurotic family, XIII-209; monster *per defectum*, XIII-268.

Discussions:

Atresia of the larynx, XII-150; acute articular rheumatism, XI-91; circulatory failure in acute infectious diseases, XIV-193; congenital cardiac malformation, XII-146, 147; cretinism, XIV-256; diarrhea, summer, XIII-49; XIV-41; enteric fever, XII-107; gruels as diluents, XI-228; a case of heart lesions, XI-207; increase of weight in children artificially fed, XI-117; intubation in diphtheria, XIV-135; methods of instruction in pediatrics, XI-23, 24, 25; milk proteids, XIV-154; peritonitis, tuberculous,

XII-26; XIV-107; renal complications of acute enteric diseases, XI-59; rheumatism, XI-91; separation of bacteria from milk, XI-39.

CRANDALL, FLOYD M.

Papers and Cases Reported:

Diarrheal diseases, II-115; heart disease, a study of 142 cases, III-73; heredity and degeneration, IX-168*j*; influenza, VIII-209; pneumonia, X-186; rheumatic children, the management of, XIV-59; rheumatism and chorea, IV-225; scurvy, a case of, in an infant of six weeks, XI-173; tetany, VII-208.

Discussions:

Constipation, V-89; diphtheria antitoxin, VIII-69; endocarditis, XIV-171; gruels, X-227; insanity, temporary, following typhoid fever, VIII-184; intubation, XIV-135; lithemia, IX-129; lymphatism, XI-68; milk, filtration of, VI-39; pediatrics, methods of instruction in, XI-17, 25; rheumatic children, the management of, XIV-71; rheumatism, acute articular, XI-90, 91; rheumatism and chorea, IV-243; rickets, VI-173; scurvy, VI-30; XI-181; urine, incontinence of, V-167; weight, increase of, in artificially-fed infants, XI-118.

DAVIS, EDWARD P.

Papers and Cases Reported:

Prenatal infection in diseases of infancy, IX-92; the treatment of hydrocephalus by craniectomy, XII-63.

Discussion:

Hydrocephalus, XII-66.

DORNING, JOHN.

Cases Reported:

Malformation of the heart, II-46; rupture of the bladder, VII-66.

Discussions:

Congenital influenza, II-208; cyclical albuminuria, XIII-109, 110; diphtheria antitoxin, VIII-68; enteric fever, XII-114; grippe, VII-143; gruels, XI-227; influenza, II-208; hydrocelphalus, XII-65; acute nephritis following influenza, XII-137, 139; perforation into trachea, XII-95, 97; scarlatina, VII-107; scurvy, XI-179; summer diarrhea, XIII-49; urticaria of the mucous membrane, XI-109; vaccinoid, XI-162.

EARLE, CHARLES WARRINGTON.

Papers and Cases Reported:

Diphtheria, necessity for prolonged rest after, I-117; emphysema, general, subcutaneous, I-56; grippe, III-170; IV-23; medication, II-198; tetany, I-153; typhoid fever, IV-112.

Discussions:

Cretinism, IV-178; diphtheria, IV-94; diphtheria, general prophylaxis in, II-147; diphtheria, personal prophylaxis in, I-116; emphysema, I-66; grippe, IV-32, 37; laryngismus stridulus, IV-

195, 199; meningitis, tuberculous, II-78; O'Dwyer's tubes, II-196; peroxid of hydrogen, IV-215; purpura, II-164, 166; stomach, capacity of, II-176; typhoid fever, IV-149.

EDSALL, DAVID L.

Discussions:

The proteids of milk, XIV-155; autointoxication with neutral magnesium phosphates in the urine, XIV-189.

ERDMANN, JOHN F.

Intussusception—surgical comments, XIV-24.

FISCHER, LOUIS.

Discussions:

Ozone, value of nascent, IV-21; rheumatism and chorea, IV-241; tracheal and bronchial casts in diphtheria, IV-93, 94; tuberculosis, anemia of, IV-173.

FORCHHEIMER, F.

Papers and Cases Reported:

Anemia, V-135; alimentary canal, elimination as an etiological factor in diseases of, VIII-220; cough in influenza simulating whooping cough, XII-152; "enanthem," the use of the term,XIV-72; eulogy on Drs. John M. Keating and Charles Warrington Earle, VI-4; a pediatric poem, VII-7; german measles, the enanthem of, X-118; mouth, nomenclature of diseases of, VI-54; pertussis, V-116; pneumonia, diagnosis of, III-18; stomatitis, aphthous, III-423; tonsillar infections, remote diseases arising from, XIV-91; vaccinoid, XI-155.

Discussions:

Constipation,VI-46; "enanthem," the use of the term, XIV-75; gastric neurosis, V-194; german measles, X-125, 128; gruels, XI-227, 228; milk, separation of bacteria from, XI-39; nephritis, III-231; X-145; pediatrics, methods of instruction in, XI-20, 21; peritonitis, tuberculous, XIV-105; pneumonia, III-55, 57; scurvy, X-25; stomatitis, III-254; tonsillar infection, XIV-101; vaccinoid, XI-162; weight, increase of, in artificially-fed infants, X-119, 120.

FREEMAN, ROWLAND G.

Papers and Cases Reported:

Liver, lesions of, XI-241; malaria, (?) a mild epidemic of atypical, XIII-225; milk, low temperature pasteurization of, VIII-113; milk, necessity of sterilization by heat, X-35; milk bottle, improved form of, IX-47; milk, separation of bacteria from, XI-37; nephritis, acute, following influenza, XII-132; pericardium, report of four cases of adherent, VIII-171.

Discussions:

Anemias, X-170; enteric fever, XII-114; gruels, XI-227; kidney, cystic, XI-130; liver lesions, XI-252; malaria, XIII-236, 237; meningitis due to typhoid bacillus, XI-153; milk, separation of

bacteria from, XI-39; milk, pasteurization of, VIII-124; milk, proteids of, XIV-156; nephritis following influenza, XII-141; sarcoma of cutis, IX-157; scurvy, X-32; summer diarrhea, XIII-47; tonsillar infection, XIV-99; scurvy, X-32; trachea, perforation of, XII-95; vaccinoid, XI-161.

FRUITNIGHT, J. HENRY.

Papers and Cases Reported:

Cerebral abscess with amnesic aphasia, XII-56; endocarditis, VIII-129; empyema, with expectoration of pus, II-28; epistaxis, significance of, IX-84; perityphlitis, III-85; rachitis, V-168; sarcoma of kidney, VII-168; scarlet fever and complications, I-95; scorbutic, membranous colitis, XI-212; scorbutus, VI-10.

Discussions:

Alcohol, II-154; cerebral abscess, XII-62; constipation, V-89; VI-46; diarrheal disease, II-125; diphtheria, III-169; V-33; VI-73; diphtheria, nasal feeding in, VIII-78; diphtheria, personal prophylaxis in, I-116; diphtheria antitoxin, VII-48; VIII-69; X-117; emphysema, I-66; empyema, I-28; enteric fever, XII-113; feeding, artificial, I-83; feeding, breast, II-100; german measles, X-127; grippe, VII-143; influenza, II-207; intussusception, X-105; insolation, VIII-206; lumbar puncture, VIII-110; malaria, I-182, 183,185; medication, II-204; meningitis, tuberculous, II-85; meningocele, cephalohydrocele, etc., II-28; nephritis, primary, III-232; nephritis, malarial, X-144; nephritis following influenza, XII-137; nephritis, scarlatinal, III-69; pancreatic digestion of casein, XII-43; proctitis, V-62; prolapsus-recti, I-158; pertussis, V-122; purpura, II-167; rachitis, V-174; VI-172, 174; respiration, noisy, I-55; sarcoma of kidney, VII-184, 185; X-185; scarlatina, III-69; VII-108; scorbutus, III-116; trachea, perforation into, XII-96; tuberculous peritonitis, XII-24; urine of healthy children, X-99; venous congestion, VI-88.

GRAHAM, EDWIN E.

Paper and Case Reported:

Congenital, cystic kidney, XI-121.

Discussions:

Enteric fever, XII-114; gruels as diluents, XI-223; separation of bacteria from milk, XI-39.

GRIFFITH, J. P. CROZER.

Papers and Cases Reported:

Antipyretics, V-64; appendicitis, XIII-141; laryngeal stenosis, XIII-272; pertussis, V-110; retro-esophageal abscess, IX-169; scurvy, X-5; transposition of viscera, XI-32; tremor, unilateral, IX-158.

Discussions:

Anemia, V-141; cereals, XIV-241; chondrodystrophy fetalis, XIV-56, 58; diphtheria antitoxin, VIII-70; diphtheria with persistent trismus and opisthotonos, XIV-219; enanthem, XIV-75; enteric fever, XII-109; german measles, X-124; grippe, VII-142; gruels, XI-226; heart, malformation of, VII-167; inani-

tion fever, VII-65; insolation, VIII-207; intussusception, XIV-28; laryngeal stenosis, XIII-276; masturbation, VIII-189; milk, pasteurization of, VIII-124; milk proteids, XIV-157; paralysis, epidemic, XII-162; pediatrics, methods of instruction in, XI-17, 26; playrooms on city roofs, XIII-224; retro-esophageal abscess, IX-179; rheumatism, XI-91; rickets, VI-171, 172; scarlatina, VII-104; scurvy, X-24, 31; XI-181; summer diarrhea, XIII-45; transposition of viscera, XI-36; tremor, unilateral, IX-164; tuberculosis, temperature in, XIII-191, 192; urine, incontinence of, V-168; urine of healthy children, X-99; weight, increase of, in artificial feeding, XI-116.

HAMILL, S. MC.

Papers and Cases Reported:

Hemorrhage, cerebral from sinus thrombosis, XIV-196; hemorrhage into the suprarenal capsule, XII-176; extreme enlargement of the spleen with anemia, XIV-76.

Discussion:

Extreme enlargement of the spleen with anemia, XIV-90.

HAND, ALFRED JR.

Paper:

Sporadic cretinism, XIV-245.

HARRINGTON, CHARLES.

Discussion:

Milk, breast, the clinical examination of, IV-270.

HATFIELD, MARCUS P.

Paper and Cases Reported:

Icterus, congenital, pernicious, I-147.

Discussion:

Erysipelas, V-204.

HOLT, L. EMMETT.

Papers and Cases Reported:

Appendicitis, VIII-229; brain, abscess of, IX-182; constipation, V-76; diphtheria antitoxin, VII-43; VIII-21; IX-32; diphtheria of the conjunctiva, XIII-294; gastro-enteric tract, nomenclature of diseases of, VI-55; hemorrhages of the newly-born, XIII-278; heart, disease of, XI-183; heart, malformation of, I-187; VII-166; hospitals for infants, X-147; inanition fever in the newly-born, VII-60; milk, breast, the clinical examination of, IV-259; meningitis complicating pneumonia, V-210; nephritis, III-223; pneumonia, temperature in, III-30; pneumonia, diagnosis of, from bronchitis, III-48; pericarditis, VIII-169; pyelitis, VI-117; stomach, capacity of, II-168; temperature, high, of three months' duration, with double, sacculated empyema, XIII-214.

Discussions:

Alcohol, II-153; cereals, XIV-242, 243; chondrodystrophy fetalis, XIV-57; diarrheal diseases, II-125; diphtheria, III-165; XIV-218; diphtheria antitoxin, VIII-68, 70; em-

pyema, I-28; IX-89; endocarditis, VIII-140; erysipelas, V-203; feeding, artificial, I-82; feeding, breast, II-99; feeding, nasal, VIII-77; gavage, III-152; gastric neurosis, V-191, 194; gastro-enteric tract, nomenclature of diseases of, VI-59, 60, 61; grippe, IV-33; VII-141, 145; heart, malformation of, III-128; hemorrhages of the newly-born, XIII-281; hernia, diaphragmatic, I-123; IX-111; hydrorrhachis, IV-106; incubators, V-46; inanition fever, VII-65; influenza, II-207; lithemia, IX-128; lumbar puncture, VIII-111; lung, abscess of, V-210; malaria, I-182, 186; masturbation, VIII-189; medication, II-203; meningitis, cerebro-spinal, VIII-201; meningitis, tuberculous, IX-82; meningitis, complicating pneumonia, V-222; milk, breast, the clinical examination of, IV-272; milk laboratories, IV-256; milk, filtration of, VI-39, 40; milk, pasteurization of, VIII-123; milk proteids, XIV-153; neuroses, VII-26; nephritis, III-232; ozone, value of nascent, IV-19; pancreatic digestion of casein, XII-43, 45; papillomata, multiple, of larynx, VIII-162; pertussis, V-121; pneumonia, III-55, 56; Pott's disease, VI-105; pyelitis, VI-133; pyloric stenosis, XIII-293; rachitis, VI-171, 174; rheumatism and chorea, IV-240, 241; sarcoma of the suprarenal capsule, VII-76; sarcoma of the kidney, VII-184; sclerema, I-146; scorbutus, III-116; X-31; stomach, capacity of, II-176, 177; summer diarrhea, XIII-46; tremor, unilateral, IX-163; tuberculosis, temperature in, XIII-192; urine of healthy children, X-109.

HOWARD, WILLIAM TRAVIS.

A case of congenital malformation of the heart, III-120.

HUBER, FRANCIS.

Papers and Cases Reported:

Cephalhematoma, ossified, VII-118; constipation, VI-41; diphtheria with tracheal and bronchial casts, IV-87; empyema, I-18; fingers, congenital hypertrophy of, VI-61; goitre, IX-168*d*; intussusception—clinical remarks, XIV-17; lung, abscess of, III-235; lung, abscess of, operation; recovery, V-206; naso-pharyngeal disease, XII-81; meningocele, spurious, cephalohydrocele, cephalocele, II-10; peritonitis, following vulvo-vaginal catarrh, simulating perforation of the appendix, I-124; pulmonary gangrene, XIII-263; rectal polypi, XIII-112; sacro-coccygeal tumor, operation; recovery, IV-200; scurvy, XI-215; scurvy and marasmus, XIII-161; teratoma of the sacrum and coccyx, X-209; urine, incontinence of, XI-163.

Discussions:

Croup membranous, I-69; cretinism, IV-178, 179; diphtheria, XIV-218; empyema, I-28; emphysema, general subcutaneous, I-65, 66; grippe, IV-33; intussusception, X-104, 105; XIV-33; laryngismus stridulus, IV-195, 198; lung, abscess of, V-210; malaria, I-183, 185; peroxid of hydrogen, IV-215; respiration, noisy, I-55; sclerema neonatorum, I-146; teratoma of the sacrum and coccyx, X-217; typhoid fever, IV-141, 143.

JACKSON, HENRY.

Papers and Cases Reported:

Nasal feeding in diphtheria, VIII-71; nephritis, chronic, in

children and young adults, III-215; obituary, Dr. J. Amory Jeffries, IV-272; purpura, a fatal case of, with notes on recent literature, II-158.

Discussions:

Broncho-stenosis, IV-187; milk laboratories, IV-255; tuberculous peritonitis, XII-27.

JACOBI, ABRAHAM.

Papers and Cases Reported:

Aneurism, I-227; asphyxia of the newly-born, II-178; dermoid cyst over large fontanel, X-129; heart, malformation of, in an adult, VII-161; hydrorrhachis, club feet, etc., IV-98; idiocy, amaurotic, family, X-41; milk sugar in infant feeding, XIII-150; peroxid of hydrogen, IV-208; pygopagus, Dr. S. Tynberg's, VII-121; relation of pediatrics to general medicine, I-6; sarcoma of the cutis, IX-150.

Discussions:

Alopecia areata, IV-44; anemias, X-169; alcohol, II-156, 157; circulatory failure in acute infectious diseases, XIV-193; chondrodystrophy fetalis, XIV-55; croup, I-69, 70; diphtheria, IV-95; diphtheria antitoxin, VII-57, 58; diphtheria, following tonsillotomy, VI-71, 73, 74; diphtheria with persistent trismus and opisthotonos, XIV-218; emphysema, I-66; empyema, IX-90; X-84; endocarditis, XIV-172; feeding, breast, II-99; gangrene following scarlet fever, VII-117; gastro-enteritis, mechanical treatment of, I-247, 248; german measles, X-125, 126; grippe, IV-32; hydrorrhachis, IV-106, 108; hyperpyrexia, VII-150; heart, malformation of, VII-168; inanition fever, VII-64; influenza, II-208; intussusception, X-106; XIV-28, 29; laryngismus stridulus, IV-196, 197, 199; lithemia, IX-128; lung, re-expansion of collapsed, I-38, 39, 41, 42; malaria, I-184, 187; meningitis, tuberculous, II-82; IX-82, 83; milk laboratories, IV-257, 258; nephritis, III-233, 234; X-146; neuroses, VII-26; peritonitis, tuberculous, XIV-107, 109; peroxid of hydrogen, IV-216; pneumonia, III-52, 53, 55, 56, 57; proctitis, V-63, 64; purpura, II-165, 166; rheumatism, XIV-68; rheumatism and chorea, IV-239; sarcoma of the cutis, IX-157; sarcoma of the kidney, VII-185; X-184; scarlatina, VII-107; sclerema, I-146; scleroderma, V-159; scorbutus, III-117, 118, 119, 120; X-29; stomach, capacity of, II-177; teratoma of sacrum and coccyx, X-217; tonsillar infection, XIV-99; tuberculosis, V-58; typhoid fever, IV-137, 138, 146, 147, 148; thymus gland, sudden death due to pressure from enlarged, XIV-194, 195.

JEFFRIES, JOHN AMORY.

Paper:

Summer diarrheas of infancy, I-249.

Discussions:

Feeding, artificial, I-84; lung, re-expansion of collapsed, I-42; malaria, I-182.

JENNINGS, CHARLES G.

Papers and Cases Reported:

Autointoxication with neutral magnesium phosphate in the

urine, XIV-184; infantile scurvy, X-5; lumbar puncture, VIII-109, scarlatiniform erythema, VII-84; temperature curve in acute croupous pneumonia, XIV-201.

Discussions:

Acute articular rheumatism, XI-90; gruels, XI-224; acute nephritis following influenza, XII-138; intubation in diphtheria, XIV-134; intubation tubes, retained, IX-63, 64; nasal feeding in diphtheria, VIII-78; rheumatism, XI-90; sarcoma of the kidney, X-184; scurvy, X-25, 30; tonsillar infection, XIV-101.

KEATING, JOHN M.

Papers and Cases Reported:

Osteitis of the hip, a case of tuberculous, III-69; tuberculosis, transmission of, to the fetus, V-47.

Discussions:

Feeding, artificial, I-82; gastro-intestinal disturbances, the mechanical treatment of, I-245; malaria, I-184.

KERLEY, CHARLES GILMORE.

Papers and Cases Reported:

Diphtheria with and without antitoxin; report of 159 cases, XIV-124; diphtheria, laryngeal, seven cases treated with antitoxin; one death, X-111; acute nephritis of malarial origin, X-140; gavage in the treatment of persistent vomiting, III-137; goitre, a case of ophthalmic, apparently cured by thyroid extract, IX-168*g*; intubation, XIV-136; summer diarrhea, a study of 555 cases, XIII-36.

Discussions:

Anemia, pernicious, XIII-90; circulatory failure, XIV-192; diphtheria, XIV-218; gavage, III-153; intubation, XIV-136; malaria, XIII-235; malarial nephritis, X-146; summer diarrhea, XIII-50, 52, 55, 57.

KILIANI, OTTO G. T.

Paper:

The filtration of milk, VI-34.

Discussion:

The filtration of milk, VI-38.

KNAPP, HERMAN.

Discussion:

Cerebral abscess, XII-61.

KOPLIK, HENRY.

Papers and Cases Reported:

Cretinism, symptomatology of, and other forms of idiocy, XIII-250; diphtheria, forms of, simulating simple angina, IV-66; diphtheria, rapid bacteriological diagnosis of, IX-44; empyema, etiology of, II-50; gastro-intestinal derangements of

infancy, management of, XII-7; hemorrhage, pulmonary, following exploratory puncture of chest, XI-27; pertussis, heart strain in, V-90; thyroids, their use in diseases other than cretinism, IX-65; stethoscope, a new binaural, XI-171; tuberculosis of the testis, I-128; weight, increase of, in artificially-fed infants, XI-110.

Discussions:

Albuminuria, cyclical, XIII-110; anemia, V-142; anemia, pernicious, XIII-88; cereals, XIV-240; diphtheria, III-163, 164, 166; V-34; VI-71; XIV-218; diphtheria antitoxin, VII-53; empyema, I-27, 28; II-33; IX-90; gastric neurosis, V-194; gastro-intestinal disturbances, mechanical treatment of, I-244, 247; gavage, III-153; gruels, XI-226; head-nodding and head-rotation in rickets, XII-79; hydrorrhachis, IV-107; hydrocephalus, XII-65; intestinal obstruction, XII-71; laryngeal stenosis, XIII-276; laryngismus stridulus, IV-196; the little finger of the Mongolian idiot, XIII-183; liver lesions, XI-252; medication, II-203; meningitis due to the typhoid bacillus, XI-153; milk laboratories, IV-254, 258; milk, preservation of, V-42; milk, separation of bacteria from, XI-39; milk proteids, XIV-155; nephritis, III-231, 232; pancreatic digestion of casein, XII-44; peritonitis following vulvo-vaginal catarrh, I-127; peritonitis, tuberculous, XII-27; XIV-106, 108; peroxid of hydrogen, IV-215; pneumonia, III-51; proctitis, V-64; purpura, II-167; renal complications of acute enteric diseases, XI-58; retro-esophageal abscess, IX-177; rickets, VI-173, 174, 175; rheumatism, XI-91; XIV-69; sclerema, I-146; V-160; scurvy, XI-181; stomatitis, III-252; summer diarrhea, XIII-45; transposition of viscera, XI-35; typhoid fever, IV-137; weight, increase of, in infants artificially fed, XI-116, 117, 118, 120.

LADD, MAYNARD.

Paper:

Pernicious anemia in infants, XIII-68.

LA FLEUR, HENRI A.

Paper:

Notes on a case of insolation, VIII-205.

Discussions:

Diphtheria antitoxin, VIII-68; a case resembling typhoid fever, VIII-243; insolation, VIII-208.

LATIMER, THOMAS L.

Papers and Cases Reported:

Paraplegia, spastic, I-195; pneumonia, diagnosis of, III-11.

Discussion:

Malaria, I-186.

LICHTENSTEIN, JACOB.

Paper:

The symptomatology of cretinism and other forms of idiocy, XIII-250.

LOCKWOOD, WILLIAM F.

Cases Reported:

Three cases of purulent otitis media, VII-129; a case of varicella gangrenosa, IX-131.

Discussion:

Purulent otitis media, VII-135.

MACDONALD, A. A.

Discussion:

Malformation of the heart, VII-167; scarlatina, VII-108.

MARTIN, C. F.

Discussion:

Extreme enlargement of the spleen, with anemia, XIV-89.

MCCOLLOM, JOHN H.

Paper:

Intubation in diphtheria, XIV-130.

Discussion:

Intubation in diphtheria, XIV-136.

MEIGS, A. V.

Paper:

Feeding, artificial, I-71.

Discussions:

Empyema, I-28; feeding, artificial, I-85.

MILLER, D. J. MILTON.

Papers and Cases Reported:

Acute articular rheumatism in infants under one year, XI-79; three cases of head-nodding and head-rotation in rachitic infants, XII-72; acute hemorrhagic nephritis following influenza, XIII-193.

Discussions:

Acute articular rheumatism, XI-90; atresia of the larynx, XII-150; endocarditis, XIV-172; enteric fever, XII-114; gruels as diluents, XI-224; head-nodding and head-rotation, XII-80; increase of weight in children artificially fed, XI-117; milk proteids, XIV-154; pancreatic digestion of casein, XII-44; perforation into trachea, XII-96; peritonitis, tuberculous, XIV-108; rheumatism, XI-90; urticaria of the mucous membranes, XI-109.

MORRILL, F. GORDON.

Papers and Cases Reported:

Immunizing effects of diphtheria antitoxin, VII-27; analysis of 100 cases of frank pneumonia, IX-144.

Discussions:

Diphtheria antitoxin, VII-59; grippe, VII-144; temperature curve in acute croupous pneumonia, XIV-204.

MORSE, J. LOVETT.

Papers and Cases Reported:

The classification of the anemias of infancy, X-161; arsenical poisoning, XIII-119; chondrodystrophy fetalis, XIV-42; heart disease, an analysis of thirty-two cases of congenital, XIII-134; infantile scurvy, X-5; renal complications of acute enteric diseases, XI-40.

Discussions:

Amaurotic family idiocy, XIII-213; anemias, X-170; XIII-88; chondrodystrophy, fetalis, XIV-55, 58; gruels as diluents, XI-223; hemorrhage of the newly-born, XIII-280; incubator baby, feeding of, XIII-132; infantile scurvy, X-25-28; increase of weight in infants artificially fed, XI-118; renal complications of acute enteric diseases, XI-59; sarcoma of the kidney, X-184; summer diarrhea, XIII-49.

NORTHRUP, WM. P.

Papers and Cases Reported:

Heart, congenital malformation of, VI-76; diphtheria antitoxin in private practice, VIII-21; diphtheria antitoxin in laryngeal cases, IX-32; larynx, atresia of, XII-148; meningitis, cerebro-spinal, VIII-198; meningitis, tuberculous, II-69; myxedema, VI-109; playrooms on city roofs, XIII-220; pediatrics, methods of instruction in, XI-7; scorbutus, III-99; sclerema, I-140; throats and ears, care of, V-129; typhoid fever in children under two years, IV-109; VII-205.

Discussions:

Anemia, pernicious, XIII-89; diphtheria, III-164, 165; diphtheria antitoxin, VIII-67, 68; endocarditis, VIII-140; empyema, IX-89, 91; enteric fever, XII-108, 115; german measles, X-125; heart lesions, VIII-169; XI-207; heart, malformation of, I-191; VIII-169; hydrorrhachis, club feet, etc., IV-107; influenza, II-207, 208; insanity, temporary, following typhoid fever, VIII-184; intussusception, X-106; intubation tubes, retained, IX-62, 64; larynx, stenosis of, VIII-233; larynx, traumatic atresia of, XII-150; lung, re-expansion of collapsed, I-34, 38, 39; malaria, XIII-235; meningitis, cerebro-spinal, VIII-202; meningitis, due to the typhoid bacillus, XI-153; meningitis, tuberculous, II-78, 87; nephritis, III-232; X-144; ozone, value of nascent, IV-20, 21; papillomata, multiple, of the larynx, VIII-162; pertussis, V-122; playrooms on city roofs, XIII-223, 224; pneumonia, III-56; renal complications of enteritis, XI-59; respiration, artificial, III-132; rickets, VI-174; sarcoma, of the cutis, IX-157; sclerema, I-147; scorbutus, III-116, 118, 119; X-24; XI-181; scleroderma, V-159; summer diarrhea, I-310; XIII-47; transformation of viscera, XI-36; typhoid fever, IV-137, 138, 139, 143, 146.

O'DWYER, JOSEPH.

Papers and Cases Reported:

Diphtheria antitoxin in private practice, VIII-21; diphtheria antitoxin in laryngeal cases, IX-32; hernia, diaphragmatic, I-119; intubation, the evolution of, VIII-9; intubation tubes,

causes and treatment of retained, IX-48; larynx, congenital stenosis of, IX-180; lung, re-expansion of collapsed, I-29; respiration, artificial, forcible, III-128.

Discussions:

Croup, membranous, I-69; gavage, III-152; hernia, diaphragmatic, IX-111; intubation tubes, causes and treatment of retained, IX-64; lung, re-expansion of collapsed, I-39, 42; respiration, artificial, III-132.

OSLER, WILLIAM.

Papers and Cases Reported:

Abdominal muscles, congenital absence of, with distended and hypertrophied urinary bladder, XIV-223; heart, enormous hypertrophy of, associated with peritonitis, ascites and pericarditis, VII-214; pneumonia, diagnosis of tuberculous, broncho, III-25; specialism, IV-6; tics or habit movements, classification of, VIII-215; tuberculosis, V-178; wry-neck, association of congenital, with marked facial asymmetry, III-133.

Discussions:

Cretinism, IV-178, 179; cardiac lesions, VIII-169; diphtheria antitoxin, VII-50, 57; VIII-68; empyema, I-27; IX-90; endocarditis, VIII-140; gangrene following scarlet fever, VII-117; heart, malformation of, VII-167; hydrorrhachis, IV-107; insanity, temporary, following typhoid fever, VIII-184; meningitis, cerebro-spinal, VIII-201. 202; milk laboratories, IV-258; pericarditis, VIII-169: peritonitis following appendicitis, VIII-231; pertussis, V-120; pyelitis, VI-132; rheumatism and chorea, IV-242; sarcoma of the kidney, VII-184; tuberculosis, anemia of, IV-174.

PACKARD, FREDERICK A.

Papers and Cases Reported:

Cretinism, sporadic, XIV-245; diphtheria antitoxin, favorable result from its use, VIII-63; endothelioma of the cerebral membranes with Jacksonian epilepsy and progressive muscular atrophy, VIII-147; urticaria of the mucous membranes, especially in connection with asthma, XI-92.

Discussions:

Cyclical albuminuria, XIII-110; heart lesions, XI-206; intussusception, XIV-27; malaria, XIII-235; malformation of the heart, VII-167; rheumatism, XIV-69, 71; urticaria of the mucous membranes, XI-109.

PEPPER, WILLIAM.

Discussion:

Double empyema, I-27.

PUTNAM, CHARLES P.

Papers and Cases Reported:

Alopecia areata, IV-39; congenital malformation of the bile duct, X-108; medicinal treatment of constipation, V-80.

Discussions:

Alopecia areata, IV-44; diphtheria, III-166, 167; empyema, I-27; gavage, III-152; intussusception, XIV-28, 31; nephritis, III-231; perityphlitis, III-98; playrooms on city roofs, XIII-223; stomatitis, aphthosa, III-253, 254; thymus gland, sudden death due to pressure from enlarged, XIV-195; vaccinoid, XI-161.

RACHFORD, B. K.

Papers and Cases Reported:

Anemia of tuberculosis, IV-151; albuminuria as a lithemic manifestation, X-85; gangrene, superficial, VIII-190; hare-lip, XIII-242; lithemia, symptomatology of, IX-112; maternal impressions, XIII-147; neuroses of childhood, VII-18; pancreatic digestion of casein, XII-29; tuberculosis, treatment of, XIII-116; urine, incontinence of, V-161; venous congestion, its influences on spinal, reflex centres, VI-81.

Discussions:

Anemia of tuberculosis, IV-174; lymphatism, XI-67; gruels, XI-225; lithemia, IX-129; maternal impressions, XIII-149; neuroses, VII-26; ozone, value of nascent, IV-22; pancreatic digestion of casein, XII-42, 44.

RAU, LEONARD S.

Paper:

Dactylitis in children, II-60.

REID, JOHN J.

Paper:

Suppurative peritonitis, II-204.

ROTCH, THOMAS M.

Papers and Cases Reported:

Anemia, pernicious, XIII-68; diphtheria of the eye, IX-135; feeding, breast, II-88; gastro-enteric tract, nomenclature of diseases of, VI-55; heart, congenital malformation of, VIII-203; iconoclasm and original thought in the study of pediatrics, III-6; incubator, a new, V-44; intussusception, X-102; loving cup presentation to, XIV-6; milk laboratories, IV-244; mouth, nomenclature of diseases of, VI-54; melanosis lenticularis progressiva, X-172; meningitis, cerebro-spinal, VII-67; X-101; osteomyelitis, multiple, XI-208; peritonitis, tuberculous, treatment of, XIV-102; rhachischisis, XII-47.

Discussions:

Alcohol, II-155; anemia, pernicious, XIII-91; cardiac lesions, VIII-169; cereals, XIV-239, 241; cretinism, IV-179; diphtheria, III-166, 169; diphtheria antitoxin, VIII-66; diphtheria and croup following tonsillotomy, VI-74; enanthem, XIV-75; enteric fever, XII-110; feeding, breast, II-100; german measles, X-126; gastric neurosis, V-192, 195; gastro-enteric tract, nomenclature of diseases of, VI-59; grippe. IV-31, 32, 37, 38; gruels, XI-224, 225, 226, 228, 229;

hemorrhages of the newly-born, XIII-280; hernia, diaphragmatic, IX-111; hydrocephalus, XII-65, 66; incubator baby, feeding of, XIII-133; intussusception, X-107; XIV-32; laryngeal stenosis, XIII-275, 276; lithemia, IX-129; little finger of the Mongolian idiot, XIII-183; lung, abscess of, V-210; meningitis, cerebro-spinal, VIII-201; meningitis, tuberculous, II-80, 86; meningitis complicating pneumonia, V-222; meningitis, due to the typhoid bacillus, XI-153; milk, breast, the clinical examination of, IV-271; milk laboratories, IV-257, 258; milk, preservation of, V-41, 43; milk, separation of bacteria from, XI-39; milk pasteurization, VIII-123; neuroses, VII-26; nephritis, III-234; nephritis, acute, following influenza, XII-138, 139, 141; nystagmus, associated with choreic movements of the head, I-239; obituary, Dr. John Amory Jeffries, IV-276; ozone, value of nascent, IV-20; pancreatic digestion of casein, XII-42, 43, 44; papillomata of larynx, VIII-162; pediatrics, methods of instruction in, XI-15, 17, 21, 24; pneumonia, III-52, 56; pericarditis, VIII-169; peritonitis, tuberculous, XII-25, 27; XIV-105, 108, 109; Pott's disease, VI-104; purpura, II-166; rachitis, V-174; retro-esophageal abscess, IX-178; scorbutus, III-116, 119; XI-181; stomach, capacity of, II-175, 176, 177; stomatitis aphthosa, III-253, 255; summer diarrhea, XIII-50, 53, 54; trachea, perforation into, XII-94, 95; tremor, unilateral, IX-162, 164; tuberculosis, anemia of, IV-174; typhoid fever, IV-147; urine, incontinence of, V-167; urine of healthy children, X-99; venous congestion, VI-88; weight, increase of, in artificially-fed infants, XI-116, 117, 118, 120.

SAUNDERS, E. W.

Paper:

Pilocarpin in the treatment of scarlet fever, XIV-181; pyloric stenosis, XIII-282.

Discussions:

Cereals, XIV-243; summer diarrhea, XIII-54.

SCHAELAU, B.

Paper:

Synopsis of 56 cases of empyema with operation, IX-87.

Discussion:

Influenza, II-208.

SCHWYZER, F.

Paper:

Diphtheria antitoxin, sudden death after its use, VII-54.

SEIBERT, AUGUST.

Papers and Cases Reported:

Alcohol, II-148; broncho-stenosis, syphilitic, IV-180; diphtheria, treatment of, III-155; diphtheria antitoxin, its value and toxic after effects, VII-46; diphtheria antitoxin, sudden death after its use, VIII-54; gastro-intestinal disturbances, mechanical treatment of, I-240; ichthyol ointment in eruptive fevers, VII-101; milk, filtration of, VI-30; scarlatina, treatment of the

nasopharynx in, XIII-64; typhoid fever, treatment of, XIII-118; typhoidal appendicitis, XIV-115.

Discussions:

Broncho-stenosis, IV-188; circulatory failure in acute infectious diseases, XIV-192; diphtheria, III-164, 165, 166, 167, 169; IV-93, 94; V-33; diphtheria antitoxin, VII-59; VIII-67, 70; diarrhea, summer, XIV-40; enanthem, XIV-75; endocarditis, XIV-173; erysipelas, V-203, 204; gastro-intestinal disturbances in infancy, mechanical treatment of, I-247, 248; gastro-enteric tract, nomenclature of diseases of, VI-59, 60; gavage, III-152; gastric neurosis, V-193; grippe, VII-144, 145; hyperpyrexia, VII-149; insolation, VIII-207; influenza, VIII-213; laryngismus, stridulus, IV-196; liver, enlargement of, with typhoid symptoms, VIII-243; malaria, I-183; medication, II-203; milk, filtration of, VI-39, 40, 41; milk, preservation of, V-41; nephritis, III-230, 234; peritonitis, tuberculous, XIV-108; peroxid of hydrogen, IV-214; pertussis, V-124; papillomata of larynx, VIII-162; pyelitis, VI-133; rickets, V-174; VI-172, 174, 175; scurvy, VI-29; stomach, capacity of, II-175; typhoid fever, IV-141; typhoidal appendicitis, XIV-119.

SHAW, W. R.

Discussion:

Empyema, II-59.

SHAW, H. L. K.

Discussion:

Appendicitis, XIII-146.

SMITH, J. LEWIS.

Papers and Cases Reported:

American Pediatric Society, its present and its future, II-6; diphtheria, true and pseudo, IV-76; diphtheria, peroxid of hydrogen in treatment of, V-17; feeding, artificial, I-86; nephritis, scarlatinal, III-201; rachitis, causes of, V-195; scarlet fever, prophylaxis in, II-129; scarlet fever, prophylaxis as to complications and sequelæ, III-58; tetanus neonatorum, VI-143; VII-192.

Discussions:

Alcohol, II-155, 157: diphtheria, III-164; heart, malformation of, III-128; influenza, II-207, 208; meningitis, V-222; meningitis, tuberculous, II-86; nephritis, III-231, 232; pertussis, V-125; pneumonia, III-55, 57; purpura, II-167; rachitis, V-174; scorbutus, III-120.

SNOW, IRVING M.

Papers and Cases Reported:

Acetanilid poisoning by absorption from the umbilicus, IX-39; diphtheria, with persistent trismus and opisthotonos—Escherich's pseudo tetanus, XIV-210; gastric neurosis, V-185; heatstroke in infants, X-132; intestinal obstruction, XII-67; meningocele, false, V-174; papillomata, multiple of larynx, VIII-159; respiratory failure in the newly-born, XIII-122; rickets,

its frequency among Neapolitan children in America, VI-160, tetanus neonatorum, a case resembling, VII-150.

Discussions:

Diphtheria with persistent trismus and opisthotonos, XIV-219; gastric neurosis, V-195; intestinal obstruction, XII-71; rickets among Neapolitan children, VI-176.

STANDISH, MYLES.

Discussion:

Diphtheria of the eye, IX-142.

STARR, LOUIS.

Papers:

Anomalous scarlatina, VII-90; proctitis in early infancy, V-58.

Discussions:

Constipation, V-89; proctitis, V-63, 64; scarlatina, VII-109.

STARR, M. ALLEN.

Discussion (letter):

Rheumatism and chorea, IV-238.

STENGEL, ALFRED.

Paper:

The blood in infancy and childhood, XII-214.

STOWELL, W. L.

Discussions:

Congenital constipation, VI-46; rickets, VI-174.

SWIFT, GEORGE MONTAGUE.

Paper:

Four cases of adherent pericardium in children, VIII-171.

THOMPSON, W. GILMAN.

Discussion (letter):

Gavage, III-153.

TOWNSEND, CHARLES W.

Papers and Cases Reported:

Cretinism, IV-175; cretin, a pregnant, VIII-234; influenza, congenital, II-205; heart disease, a study of thirty cases of congenital, XI-72; hemorrhagic disease of the new-born, VI-47; incubator baby, feeding of, XIII-129; rheumatism and chorea, IV-217; thigh-friction in infants, VIII-186; rickets, congenital, VI-106; summer diarrhea, II-101.

Discussions:

Anemia, pernicious, XIII-90; diphtheria antitoxin, VIII-69; chorea and rheumatism, IV-240, 241; cretinism, IV-179; incubator baby, feeding of, XIII-133; influenza, II-208; milk proteids, XIV-155; meningitis, cerebro-spinal, VIII-201; rheumatism and chorea, IV-240, 241; summer diarrhea, XIII-54; thigh friction, VIII-189.

TYSON, JAMES.

Discussion:

The urine of healthy children, X-99.

VAUGHAN, VICTOR C.

Paper:

A chemical study of summer diarrhea in infants, II-109.

Discussion:

Alcohol, II-154, 155, 156.

VINEBERG, HIRAM N.

Paper:

Diagnosis and treatment of malaria in children, I-159.

Discussions:

Empyema, I-28; croup, I-70; gastro-intestinal disturbances in infants, I-247; hernia, diaphragmatic, I-124; malaria, I-185.

WARNER, FREDERIC M.

Paper:

A case of sarcoma of the kidney, II-65.

WATSON, WILLIAM PERRY.

Discussions:

Hyperpyrexia, VII-149; malaria, I-186.

WENTWORTH, A. H.

Papers and Cases Reported:

Lumbar puncture, VIII-85; meningitis due to the typhoid bacillus, XI-145.

Discussions:

Anemia, with enlarged spleen, XIV-89; anemia, pernicious, XIII-87, 89; cardiac lesions, VIII-170; diphtheria antitoxin, VIII-69; influenza, XI-239, 240; insolation, VIII-208; lesions of the liver, XI-252; lumbar puncture, VIII-111; XI-239; malaria, XIII-236; maternal impressions, XIII-149; meningitis, cerebro-spinal, VIII-201; meningitis, due to the typhoid bacillus, XI-153; milk proteids, XIV-157; pericarditis, VIII-170; scurvy, XI-181; summer diarrhea, XIII-53; thigh-friction, VIII-189; tuberculosis, temperature in, XIII-191; urticaria of the mucous membranes, XI-109; weight, increase of, in children artifically fed, XI-118, 119.

WESTCOTT, THOMPSON S.

Papers:

Cerebro-spinal symptoms in influenza, XI-230; the management of fat percentages in difficult cases of feeding, XIV-137.

Discussions:

Cerebro-spinal symptoms in influenza, XI-240; milk proteids, XIV-152, 157.

WEST, J. PARK.

Papers:

Enlarged bronchial nodes a manifestation of lymphatism.

XI-61; the little finger of the mongolian idiot and of normal children, XIII-177.

Discussion:

Perforation into the trachea, XII-96, 97.

WHITE, JAMES C.

Discussion:

Alopecia areata, IV-43.

WHITE, C. Y.

Paper:

The blood in infancy and childhood, XII-214.

WILSON, J. C.

Papers and Cases Reported:

Symmetrical gangrene following scarlet fever, VII-109; *tic convulsif*, IX-165.

Discussions:

Diphtheria antitoxin, VIII-68; endocarditis, XIV-171; empyema, XIII-219; enanthem, XIV-75; enteric fever. XII-111; hemorrhages of the newly-born, XIII-280; rheumatism, XIV-69; scarlatina, VII-107; temporary insanity following typhoid fever, VIII-184.

WILLIAMS, HAROLD.

Paper:

A case of measles complicated by appendicitis, XIII-254.

Discussion:

Summer diarrhea, XIII-54.

WINTERS, JOSEPH E.

Paper:

The use of alcohol in the treatment of diseases of children, II-152.

Discussions:

Anemia, pernicious, XIII-90; albuminuria, cyclical, XIII-110; cereals, XIV-243; diphtheria antitoxin, VII-54; empyema, X-84; feeding, artificial, I-83; intussusception, X-105; lung, abscess of, V-210; meningitis, tuberculous, II-84; pertussis, V-123; playrooms on city roofs, XIII-223, 224; rachitis, V-174; scurvy, X-28; summer diarrhea, XIII-47, 52, 53; typhoid fever, IV-139.

WOODWARD, W. C.

Discussions:

Diphtheria antitoxin, VII-53; grippe, VII-145.

YALE, LEROY M.

Papers:

Address in presenting loving cup to Dr. Rotch, XIV-6; local treatment of constipation, V-85.

Discussions:

Diphtheria antitoxin, VIII-68; thigh-friction in infants, VIII-189.

Index by Subjects.

Abdominal muscles, congenital abscence of, with distended and hypertrophied urinary bladder....Osler, XIV-223

Abscess, cerebral, with amnesic aphasia....Fruitnight, XII-56

Abscess of the brain, with malformation of the heart....Northrup, VI-76

Abscess of the brain, tuberculous....Adams, VIII-126

Abscess of the brain, report of five cases and summary of twenty-seven others....Holt, IX-182

Abscess of the lung....Huber, III-235; V-206

Abscess, retro-esophageal....Griffith, IX-169

Acetanilid poisoning by absorption from umbilicus....Snow, IX-39

Ascites, recurring, chronic peritonitis, hypertrophy of the heart with adherent pericardium....Osler, VII-214

Alcohol, its use in pediatrics....Seibert, II-148; Winters, II-152

Discussion.—Holt, 153; Fruitnight, 154; Vaughan, 154, 155, 156; Smith, 155, 157; Chapin, 155, 156; Rotch, 155; Jacobi, 156, 157.

Albuminuria as a lithemic manifestation....Rachford, X-85

Albuminuria, cyclical....Churchill, XIII-93

Discussion. — Dorning, 109, 110; Churchill, Packard, Winters, Koplik, 110.

Alimentary canal, etiology of diseases of....Forchheimer, VIII-220

Alopecia areata, an epidemic of....Putnam, IV-39

Discussion.—Bowen, 42; White, 43; Jacobi, Putnam, 44.

American Pediatric Society, a review of the work of....Blackader, V-8

American Pediatric Society, constitution and by-laws of....Original, I-IX; Revised, XI-253

American Pediatic Society, its object....Smith, II-6

Amaurotic family idiocy....Jacobi, X-41; Cotton, XIII-209

Anemia, a case of, with extreme enlargement of the spleen....Hamill, XIV-76

Anemia, pernicious, in infants....Rotch and Ladd, XIII-68

Discussion.—Wentworth, 87, 89; Koplik, Morse, 88; Northrup, 89; Churchill, Kerley, Townsend, Winters, 90; Rotch, 91.

Anemia, treatment of ..... Forchheimer, V-135

Discussion.—Caillé, Griffith, 141; Koplik, 142.

Anemia of tuberculosis..... Rachford, IV-151
Anemias of infancy, classification of..... Morse, X-161

Discussion.—Jacobi, 169; Freeman, Morse, 170.

Aneurism ..... Jacobi, I-227
Angina, simple, forms of diphtheria simulating..... Koplik, IV-66
Angina, the relation of pseudo-diphtheritic (scarlatinal) to diphtheria..... Booker, IV-45
Antipyretic drugs..... Griffith, V-64

Discussion.—Christopher, 75.

Anuria ..... Cotton, XI-137
Anuria and endocarditis, with cardiac malformation ..... Cotton, XII-142
Aphasia, amnesic, with cerebral abscess..... Fruitnight, XII-56
Aphasia, amnesic, scarlatina anginosa, pneumonia crouposa ..... Adams, VII-136
Appendicitis in children of two years and under ..... Griffith, XIII-141

Discussion.—Shaw, 146.

Appendicitis..... Holt, VIII-229
Appendicitis, a case of, complicating measles..... Williams, XIII-254
Appendicitis, a case of peritonitis simulating, following vulvo-vaginal catarrh..... Huber, I-124
Appendicitis, typhoidal..... Seibert, XIV-115
Arsenical poisoning..... Morse, XIII-119
Arteriosclerosis, a case of..... Baines, XIII-58
Arthritis, multiple, a case of gonorrheal..... Acker, XIII-238
Asphyxia of the newly-born, results of..... Jacobi, II-178
Asthma associated with urticaria of the mucous membranes ..... Packard, XI-92
Asthma dyspepticum, simulated by recurrent attacks due to diaphragmatic hernia..... Booker, IX-100
Ataxia, a case of..... Blackader, I-191
Autointoxication with neutral magnesium phosphate in the urine..... Jennings, XIV-184

Discussion.—Edsall, Christopher, 189.

Bacteria, separation of, from milk..... Freeman, XI-37
Bacteria of summer diarrhea..... Booker, I-198
Bacteriology of summer diarrhea..... Jeffries, I-249
Brain, abscess of..... Holt, IX-182
Brain, abscess of, with congenital malformation of the heart..... Northrup, VI-76
Brain, abscess of, tuberculous..... Adams, VIII-126
Brain, multiple tumor of..... Chapin, VIII-236
Bile duct, congenital malformation of..... Putnam, X-108
Bladder, rupture of..... Dorning, VII-66
Bladder, stone in, followed by prolapsus-recti..... Caillé, I-157

Blood in infancy and childhood, with reports of blood examinations in acute rheumatism, bronchitis, cerebral palsy, convulsions, eczema, enteritis, epilepsy, heart disease, meningitis, noma, pertussis, pleural effusion, pneumonia, rachitis, tuberculous caries with cold abscess, typhoid fever, varicella.....Stengel and White, XII-214
Bottle, improved form for pasteurizing and nursing.....Freeman, IX-47
Breast milk, clinical examination of.....Holt, IV-259

Discussion.—Harrington, 270; Rotch, 271; Blackader, Holt, 272.

Bronchitis, diagnosis from broncho-pneumonia.....Holt, III-48
Broncho-stenosis, syphilitic.....Seibert, IV-180

Discussion.—Caillé, Jackson, 187; Seibert, 188.

Bronchial nodes, enlarged, a manifestation of lymphatism.....West, XI-61

Discussion.—Rachford, 67; Crandall, Chapin, 68.

Bronchial nodes, perforation of tuberculous into trachea.....Caillé, XII-94
Carbolic acid poisoning.....Adams, VI-134
Cardiac (see Heart).
Cardiac anomaly, a case of.....Acker, VII-155
Cardiac inflammations in children, importance of prolonged rest after.....Holt, XI-183
Carpo-pedal contractions a manifestation of tetany.....Earle, I-153
Casein, pancreatic digestion of.....Rachford, XII-29
Cephalocele, acquired, traumatic cephalohydrocele, spurious meningocele.....Huber, II-10
Cephalhematoma, ossified.....Huber, VII-118
Cereals, the place of, in infant feeding.....Chapin, XIV-229

Discussion.—Rotch, 239, 241; Koplik, 240; Griffith, 241; Holt, 242, 243; Winters, Saunders, 243; Chapin, 244.

Cerebellum, sarcoma of.....Adams, XI-69
Cerebral hemorrhage from sinus thrombosis in an infant with sigmoid fusion of the kidneys.....Hamill, XIV-196
Cerebral abscess with amnesic aphasia.....Fruitnight, XII-56

Discussion.—Knapp, 61; Chapin, Fruitnight, 62.

Cerebro-spinal symptoms in influenza.....Westcott, XI-230
Chorea and rheumatism,
Townsend, IV-217; Crandall, IV-225; Adams, IV-231
Chondrodystrophy fetalis, a case of.....Morse, XIV-42

Discussion.—Jacobi, 55; Griffith, 56,

58; Holt, 57; Christopher, 57, 58; Morse, 55, 58.

Circulatory failure in acute infectious disease.....Caillé, XIV-190
Discussion.—Acker, 191, 193; Blackader, Caillé, 191; Kerley, Seibert, 192; Jacobi, Cotton, Adams, 193.

Clinical diagnosis chart and record.....Caillé, XI-142
Club feet, congenital luxation of femora and hydrorrhachis .....Jacobi, IV-98
Colitis (see Diarrhea).
Colitis, scorbutic, membranous.....Fruitnight, XI-212
Coma, malarial.....Acker, XII-163
Constipation, congenital.....Huber, VI-41
Discussion.—Stowell, Fruitnight, Forchheimer, 46.

Constipation, dietetic treatment of.....Holt, V-76
Constipation, local treatment of.....Yale, V-85
Constipation, medicinal treatment of.....Putnam, V-80
Discussion.—Starr, Fruitnight, Crandall, 89.

Craniectomy in hydrocephalus.....Davis, XII-63
Cretin, a pregnant.....Townsend, VIII-234
Cretinism, sporadic.....Packard and Hand, XIV-245
Discussion.—Cotton, 256.

Cretinism, sporadic congenital.....Townsend, IV-175
Discussion.—Huber, Osler, 178, 179; Earle, 178; Rotch, Townsend, 179.

Cretinism, the use of thyroids in diseases other than.....Koplik, IX-65
Cretinism, symtomatology of and other forms of idiocy.....Koplik and Lichtenstein, XIII-259
Croup, membranous; tracheotomy and recovery.....Caillé, I-67
Discussion.—Jacobi, 69, 70; O'Dwyer, Huber, Brown, 69; Vineberg, 70.

Cutis, sarcoma of, in the newly-born.....Jacobi, IX-150
Cystic kidney, congenital.....Graham and Coplin, XI-121
Dactylitis.....Rau, II-60
Degeneration and heredity.....Crandall, IX-168*j*
Dermoid cyst over large fontanel.....Jacobi, X-129
Diapers, the effect of tight.....Cotton, XIV-174
Diaphragmatic hernia with recurrent attacks simulating asthma.....Booker, IX-100
Discussion.—Holt, O'Dwyer, Rotch, Booker, 111.

Diarrhea (see summer diarrhea).
Diarrheal disease, etiology and treatment of.....Crandall, II-115
Discussion.—Booker, 122; Fruitnight, Holt, 125; Blackader, 128.

Diarrheal disease, a study of.....Jeffries, I-249

Discussion.—Northrup, 310; Booker, 311.

Diarrhea, summer, a bacteriological study of.....Booker, I-198
Diarrhea, summer, a chemical study of.....Vaughan, II-109

Discussion.—Booker, 122; Fruitnight, Holt, 125; Blackader, 128.

Diarrhea, summer, a study of 555 cases.....Kerley, XIII-36

Discussion. — Griffith, Koplik, 45; Buckingham, Holt, 46; Northrup, Chapin, Freeman, 47; Winters, 47, 52, 53; Adams, 48, 57; Cotton, Morse, Dorning, 49; Rotch, 50, 53, 54; Wentworth, 53; Saunders, Townsend, Williams, 54; Kerley, 50, 52, 54, 55, 57.

Diarrhea, summer, local variations in the mortality of.....Chapin, XIV-34

Discussion.—Seibert, 40; Chapin, Adams, Cotton, 41.

Diarrhea, summer, the early history of.....Booker, XIII-7
Diarrhea, summer, the treatment of.....Townsend, II-101
Diphtheria, diagnosis from pseudo-diphtheritic angina (scarlatinal).....Booker, IV-45
Diphtheria, diagnosis from simple angina.....Koplik, IV-66
Diphtheria, diagnosis, rapid bacteriological.....Koplik, IX-44
Diphtheira, etiology of true and pseudo.....Smith, IV-76
Diphtheria, etiology of pseudo-membranous; general distribution of diphtheria bacilli.....Booker, V-24

Discussion.—Seibert, Fruitnight, 33; Caillé, Koplik, 34.

Diphtheria, cases resembling —the diphtheria bacilli absent.....Buckingham, VII-186
Diphtheria with persistent trismus and opisthotonos—Escherich's pseudo-tetanus.....Snow, XIV-210

Discussion. — Jacobi, Koplik, Holt, Huber, Kerley, 218; Griffith, Snow, 219.

Diphtheria and croup following tonsillotomy, a case of.....Caillé, VI-66
Diphtheria of the eye.....Holt, XIII-294; Rotch, IX-135

Discussion.—Myles Standish, IX-142.

Diphtheria, nasal feeding in.....Jackson, VIII-71

Discussion.—Holt, 77; Booker, Fruitnight, Jennings, 78.

Diphtheria, following tracheotomy for multiple papillomata of the larynx, a fatal case.....Snow, VIII-159

Discussion.—Northrup, Holt, Rotch, Caillé, Seibert, 162.

Diphtheria, prophylaxis, general.....Caillé, II-136
Discussion.—Earle, 147.
Diphtheria, prophylaxis, personal.....Caillé, I-108
Discussion.—Fruitnight, Earle, 116.
Diphtheria, treatment by hydrogen peroxid.....Smith, V-17
Diphtheria, treatment by intubation.....McCollom, XIV-130
Discussion.—Caillé, 133; Jennings, 134; Chapin, 134, 136; Buckingham, Cotton, Crandall, 135; McCollom, Adams, Kerley, 136.
Diphtheria, treatment, submembranous, local.....Seibert, III-155
Discussion.—Koplik, 163, 164, 166; Northrup, 164, 165; Smith, 164; Holt, 165; Rotch, 166, 169: Putnam, 166, 167; Fruitnight, 169; Seibert, 164, 165, 166, 167, 169.
Diphtheria, prolonged rest necessary after some attacks.....Earle, I-117
Diphtheria with tracheal and bronchial casts.....Huber, IV-87
Discussion.—Caillé, 92; Seibert, Fischer, 93, 94; Earle, 94; Jacobi, 95.
Diphtheria antitoxin, immunizing effects of.....Morrill, VII-27
Diphtheria antitoxin, its use for immunization.....Holt, VII-43
Diptheria antitoxin, personal experience with.....Caillé, VII-38
Diphtheria antitoxin, its value and toxic after effects.....Seibert, VII-46
Diphtheria antitoxin, sudden death following its use.....Seibert and Schwyzer, VIII-54
Diphtheria antitoxin, favorable result from its use.....Packard, VIII-63
Diphtheria antitoxin, its use in private practice; report on Collective Investigations.....Committee: Holt, Northrup, O'Dwyer, Adams, VIII-21
Diphtheria antitoxin, its use in District of Columbia.....Adams, VIII-46
Diphtheria antitoxin, its use in laryngeal cases in private practice; report on Collective Investigation.
Committee: Northrup, O'Dwyer, Holt, Adams, IX-32
Diphtheria antitoxin, its effect in laryngeal diphtheria; 7 cases, 1 death.....Kerley, X-111
Diphtheria antitoxin, report of 159 cases with and without.....Kerley, XIV-124
Diphtheria antitoxin, general discussion on Adams, VIII-68; Booker, VII-52; VIII-68; Brown, IX-38; Caillé, VIII-66, 70; X-117; XIV-133; Chapin, VII-54; Crandall, VIII-69; Dorning, VIII-68; Fruitnight, VII-48; VIII-69; X-117; Griffith, VIII-70; Holt, VIII-68, 70; Jacobi, VII-57, 58; Jennings, XIV-134; Koplik, VII-53; La

Fleur, VIII-68; Morrill, VII-59; Osler, VII-50, 57; VIII-68; Northrup, VIII-67, 68; Rotch, VIII-66; Seibert, VII-59; VIII-67, 70; Townsend, VIII-69; Wentworth, VIII-69; Wilson, VIII-68; Winters, VII-54; Woodward, VII-53; Yale, VIII-68.

Dysentery, ulcerative, catarrhal, a case of.....Booker, III-192

Earle, Dr. Charles Warrington, eulogy on.....Forchheimer, VI-7

Ears and throat, the care of.....Northrup, V-129

Eczema, rapid absorption of carbolic acid by skin in.....Adams, VI-134

Effusion, diagnosis of from consolidation of the lung.....Forchheimer, III-18

Elimination as an etiological factor of diseases of the alimentary canal.....Forchheimer, VIII-220

Emphysema, general subcutaneous.....Earle, I-56; Cotton, XII-128

Discussion.—Huber, I-65, 66; Fruitnight, Jacobi, Carr, Earle, I-66.

Empyema (see Pyothorax).

Empyema, double.....Huber, I-18

Discussion.—Caillé, 26; Koplik, 27; 28; Pepper, Osler, Putnam, Carr, 27; Holt, Fruitnight, Vineberg, Meigs, Huber, 28.

Empyema, double, sacculated, prolonged high temperature in.....Holt, XIII-214

Discussion.—Chapin, 218; Wilson, 219.

Empyema, etiology of.....Koplik, II-50

Discussion.—Booker, Shaw, 59.

Empyema, synopsis of 56 cases with operation.....Scharlau, IX-87

Discussion.—Chapin, Holt, 89; Northrup, 89, 91; Osler, Jacobi, Koplik, 90.

Empyema, hemorrhage (pulmonary), following exploratory puncture of chest in supposed cases of.....Koplik, XI-27

Empyema, irrigation by submersion in.....Adams, X-80

Discussion.—Winters, Caillé, Jacobi, Adams, 84.

Empyema, with expectoration of pus, a case of.....Fruitnight, II-28

Discussion.—Carr, 32; Koplik, 33.

"Enanthem," the use of the term.....Forchheimer, XIV-72

Discussion.—Caillé, 74; Griffith, Wilson, Rotch, Seibert, Christopher, Forchheimer, 75.

Endocarditis (see Cardiac Inflammation).

Endocarditis, malignant.....Fruitnight, VIII-129

Discussion.—Northrup, Holt, Osler, 140.

Endocarditis, septic.....Adams, XIV-160

Discussion.—Crandall, Wilson, 171;

Miller, Jacobi, 172; Seibert, Adams, 173.

Endocarditis, with cardiac malformation and anuria .....Cotton, XII-142
Endothelioma of the cerebral membranes with Jacksonian epilepsy and progressive muscular atrophy.....Packard, VIII-147
Enteritis (see Diarrhea and Gastro-intestinal disease).
Enteric diseases, renal complications of.....Morse, XI-40
Enteric fever.....Blackader, XII-98

Discussion.—Blackader, 107, 116; Cotton, Adams, 107; Northrup, 108, 115; Griffith, 109; Rotch, 110; Wilson, 111; Fruitnight, 113; Graham, Freeman, Miller, Dorning, 114.

Enuresis (see Incontinence of Urine).
Epilepsy, Jacksonian, with endothelioma of the cerebral membranes and progressive muscular atrophy.....Packard, VIII-147
Epistaxis, its significance in childhood.....Fruitnight, IX-84
Eruptive fevers, ichthyol ointment in.....Seibert, VII-101
Erysipelas of the face and scalp.....Adams, V-199

Discussion.—Holt, 203; Seibert, 203, 204; Hatfield, Adams, 204.

Eulogy on Drs. John M. Keating and Charles Warrington Earle.....Forchheimer, VI-4
Eye, diphtheria of.....Rotch, IX-135
Facial asymmetry, marked, associated with wry-neck.....Osler, III-133
Feeding, artificial.....Smith, I-86; Meigs, I-71

Discussion.—Holt, Keating, 82; Fruitnight, Booker, Winters, 83; Adams, Caillé, Jeffries, 84; Meigs, 85.

Feeding, artificial, the management of fat percentages in difficult cases of.....Westcott, XIV-137
Feeding, artificial, gruels as diluents of cow's milk in.....Chapin, XI-219
Feeding, artificial, milk sugar in.....Jacobi, XIII-150
Feeding, artificial, the heating of milk for.....Freeman, X-35
Feeding, artificial, the increase of weight in.....Koplik, XI-110

Discussion.—Griffith, 116; Koplik, Rotch, 116, 117, 118, 120; Cotton, Miller, 117; Adams, Crandall, Morse, 118; Wentworth, 118, 119; Forchheimer, 119, 120; Chapin, Blackader, 119.

Feeding, artificial, the proteids of milk in.....Chapin, XIV-149
Feeding, breast, the management of difficult cases of.....Rotch, II-88
Femora, congenital luxation of, associated with club feet and hydrorrhachis.....Jacobi, IV-98

Fingers, congenital hypertrophy of.....Huber, VI-61
Finger, little, of Mongolian idiot and of normal children.....West, XIII-177

Discussion.—Rotch, Koplik, 183.

Gangrene in a case of varicella.....Lockwood, IX-131
Gangrene of the lungs complicating typhoid fever.....Acker, VIII-141
Gangrene, pulmonary.....Carr, XIII-256; Huber, XIII-263
Gangrene, superficial.....Rachford, VIII-190
Gangrene, symmetrical, following scarlet fever.....Wilson, VII-109

Discussion.—Jacobi, Osler, 117.

Gastro-intestinal derangements, management of.....Koplik, XII-7
Gastro-intestinal derangements, mechanical treatment of.....Seibert, I-240

Discussion.—Koplik, 244, 247; Keating, 245; Booker, 246; Vineberg, 247; Jacobi, Seibert, 247, 248; Caillé, 249.

Gastro-enteric tract, nomenclature of diseases of.....Rotch and Holt, VI-55
Gastric neurosis in childhood.....Snow, V-185

Discussion.—Holt, 191, 194; Christopher, Rotch, 192, 195; Seibert, 193; Koplik, Forchheimer, Caillé, 194; Snow, 195.

Gavage, in the treatment of persistent vomiting.....Kerley, III-137

Discussion.—Holt, O'Dwyer, Putnam, Seibert, 152; Koplik, Kerley, Thompson, 153.

German measles, the enanthem of.....Forchheimer, X-118

Discussion.—Griffith, 124; Northrup, Buckingham, 125; Jacobi, 125, 126; Adams, Rotch, 126; Caillé, Fruitnight, 127; Forchheimer, 125, 128.

Goitre, cured by thyroid extract.....Huber, IX-168*d*; Kerley, IX-168*g*
Grippe (see Influenza).
Grippe, manifestations of, in children.....Earle, III-170; IV 23

Discussion.—Rotch, IV-31, 32, 37, 38; Jacobi, 32; Caillé, Huber, Holt, 33; Adams, 34; Blackader, Booker, 36; Earle, 32, 37.

Grippe, a general discussion of the epidemic of 1894-1895.

VII.—Holt, 141, 145; Griffith, 142; Fruitnight, Dorning, 143; Morrill, 144; Blackader, 144, 145, 146; Seibert, 144, 145; Caillé, Buckingham, Woodward, 145.

Gruels as diluents for cow's milk.....Chapin, XI-219

Discussion.—Graham, Morse, 223; Miller, Jennings, 224; Blackader, 224, 225; Rotch, 224, 225, 226, 228, 229; Rachford, 225; Griffith, Koplik, 226; Chapin, 224, 229; Crandall, Freeman, Dorning, 227; Forchheimer, 227, 228; Cotton, 228.

Guiacol in the treatment of tuberculosis.....Rachford, XIII-166
Habit spasms (tics), classification of.....Osler, VIII-215
Hare-lip.....Rachford, XIII-242
Head-nodding and head-rotation in rickets.....Miller, XII-72

Discussion.—Koplik, 79; Miller, 80.

Heart (see Cardiac).
Heart, congenital malformation of.....Holt, I-187

Discussion.—Northrup, 191.

Heart, congenital malformation of, a case of.....Howard, III-120

Discussion.—Holt, Smith, 128.

Heart, congenital malformation of.....Rotch, VIII-203
Heart, congenital malformation of, a case of, with endocarditis and anuria.....Cotton, XII-142

Discussion.—Adams, Cotton, 146, 147; Blackader, 147.

Heart, malformation of.....Caillé, VIII-168
Heart, malformation of.....Acker, VII-154; Holt, VII-166

Discussion.—Packard, Macdonald, Griffith, Osler, 167; Jacobi, 168.

Heart, malformation of, in an adult.....Jacobi, VII-161
Heart, malformation of, with abscess of the brain.....Northrup, VI-76
Heart, malformation of, with transposition of viscera.....Griffith, XI-32

Discussion.—Chapin, Koplik, 35; Northrup, Griffith, 36.

Heart, malformation of, with transposition of great vessels.....Dorning, II-46
Heart, organic disease of, following scarlet fever.....Acker, VIII-162
Heart disease, a clinical study of 142 cases.....Crandall, III-73
Heart disease, a study of thirty cases of congenital.....Townsend, XI-72
Heart disease, an analysis of thirty-two cases of congenital.....Morse, XIII-134
Heart strain in pertussis.....Koplik, V-90
Heart lesions, discussion of, Northrup, Rotch, Olser, VIII-169; Caillé, Wentworth, VIII-170
Heart lesions, a case of.....Cotton, XI-196

Discussion.—Packard, 206; Northrup, Cotton, 207.

Heart hypertrophy, chronic peritonitis, recurring ascites and adherent pericardium.....Osler, VII-214
Heatstroke (see Insolation).
Heatstroke in infants.....Snow, X-132
Hemoptysis, fatal.....Acker, X-89; Acker, X-137
Hemorrhagic disease of the newly-born.....Townsend, VI-47
Hemorrhage, cerebral, from sinus thrombosis, with sigmoid fusion of the kidneys.....Hamill, XIV-196
Hemorrhage, gastro-intestinal, in the newly-born; treatment by suprarenal extract.....Holt, XIII-278
Discussion.—Morse, Rotch, Wilson, 280; Chapin, Carr, Holt, 281.
Hemorrhage into the suprarenal capsule.....Hamill, XII-176
Hemorrhage, pulmonary, following exploratory puncture of the chest.....Koplik, XI-27
Hernia, congenital diaphragmatic.....Booker IX-100
Discussion.—Holt, O'Dwyer, Rotch, Booker, 111.
Hernia, diaphragmatic; operation.....O'Dwyer, I-119
Discussion.—Holt, 123; Vineberg, 124.
Heredity and degeneration.....Crandall, IX-168*j*
Hip, tuberculous osteitis of the.....Keating, III-69
Hospitals for infants, their scope and limitations .....Holt, X-147
Hydrocephalus following cerebro-spinal meningitis.....Northrup, VIII-198
Hydrocephalus, treatment of, by craniectomy.....Davis, XII-63
Discussion.—Rotch, 65, 66; Dorning, Koplik, 65; Davis, 66.
Hydrorrhachis, club feet, congenital luxation of femora.....Jacobi, IV-98
Discussion.—Holt, 106; Jacobi, 106, 108; Osler, Koplik, Caillé, Northrup, 107.
Hyperpyrexia, cases of.....Chapin, VII-146
Discussion.—Seibert, Caillé, Watson, 149; Jacobi, Chapin, 150.
Ichthyol ointment in eruptive fevers.....Seibert, VII-101
Icterus, congenital pernicious, two fatal cases.....Hatfield, I-147
Idiocy, amaurotic, three cases of.....Jacobi, X-41
Idiocy, amaurotic family.....Cotton, XIII-209
Discussion.—Morse, 213.
Idiocy, cretinism and other forms of..Koplik and Lichtenstein, XIII-250
Inanition fever in the newly-born.....Holt, VII-60
Discussion.—Christopher, Jacobi, 64; Griffith, Booker, Holt, 65.
Incontinence of urine.....Huber, XI-163
Incontinence of urine, etiology of.....Rachford, V-161
Discussion.—Rotch, Crandall, Adams, 167; Griffith, 168.

Incubator baby, the feeding of an.....Townsend, XIII-129
Discussion. — Chapin, Morse, 132; Rotch, Townsend, 133.

Incubator, description of a new.....Rotch, V-44
Infancy, prenatal infection in diseases of.....Davis, IX-92
Infectious disease, acute, circulatory failure in.....Caillé, XIV-190
Influenza (see Grippe).
Influenza, congenital, a case of.....Townsend, II-205
Discussion.—Smith, Northrup, 207, 208; Blackader, Fruitnight, Holt, 207; Scharlau, Dorning, Townsend, Jacobi, 208.

Influenza, followed by acute nephritis.....Freeman, XII-132
Influenza, complicated by acute hemorrhagic nephritis.....Miller, XIII-192
Influenza, cerebro-spinal symptoms in.....Westcott, XI-230
Discussion.—Wentworth, 239, 240; Chapin, 239; Westcott, 240.

Influenza, with cough simulating pertussis.....Forchheimer, XII-152
Influenza, with report of local epidemics.....Crandall, VIII-209
Discussion.—Seibert, 213.

Insanity, temporary, following typhoid fever.....Adams, VIII-177
Discussion.—Crandall, Northrup, Wilson, Osler, 184; Adams, Christopher, 185.

Insolation, a case of, in an infant.....La Fleur, VIII-205
Discussion.—Fruitnight, 206; Seibert, Griffith, 207; Adams, La Fleur, Wentworth, 208.

Intestinal obstruction (see Intussusception).
Intestinal obstruction, symptoms of.....Chapin, VI-63
Intestinal obstruction, a case of.....Snow, XII-67
Discussion.—Caillé, Adams, Christopher, 70; Carr, Koplik, Snow, 71.

Intubation in diphtheria.....McCollom, XIV-130
Discussion.—Caillé, 133; Jennings, 134; Chapin, 134, 136; Buckingham, Cotton, Crandall, 135; Adams, Kerley, McCollom, 136.

Intubation, faulty, resulting in atresia of the larynx.....Northrup, XII-148
Intubation, the evolution of.....O'Dwyer, VIII-9
Intubation, report of 350 cases.....Brown, II-182
Discussion.—Earle, 196; Caillé, 197.

Intubation tubes, retained.....O'Dwyer, IX-48
Discussion.—Northrup, 62, 64; Jennings, 63, 64; O'Dwyer, 64.

Intussusception .....Rotch, X-102

Discussion.—Huber, 104, 105; Fruitnight, Winters, 105; Northrup, Conner, Jacobi, 106; Caillé, Rotch, 107.

Intussusception.....Huber, XIV-17; Erdmann, XIV-24

Discussion. — Caillé, Packard, 27; Jacobi, 28, 29; Putnam, 28, 31; Griffith, 28; Chapin, 29; Adams, 30, 31; Christopher, Blackader, Rotch, 32; Huber, 33.

Janiceps asymmetros, a case of.....Caillé, I-70
Jeffries, Dr. John Amory, obituary.....Jackson, IV-272

Discussion.—Rotch, 276.

Keating, Drs. John M. and Charles Warrington Earle, eulogy on.....Forchheimer, VI-4
Kidneys, congenital cystic.....Graham and Coplin, XI-121

Discussion.—Freeman, 130.

Kidneys, sigmoid fusion of, in an infant with cerebral hemorrhage.....Hamill, XIV-196
Kidney (see Renal).
Kidney, sarcoma of.....Warner, II-65
Kidney, sarcoma of.....Churchill, X-178

Discussion.—Morse, Jennings, Jacobi, 184; Fruitnight, 185.

Kidney, sarcoma of, treated with erysipelas toxin .....Fruitnight, VII-168

Discussion.—Holt, Osler, 184; Fruitnight, 184, 185; Jacobi, 185.

Larynx, cicatricial stenosis of.....Caillé, VIII-232

Discussion.—Northrup, Caillé, 233.

Larynx, congenital stenosis of, cicatricial.....O'Dwyer, IX-180
Larynx, multiple papillomata of, with tracheotomy, followed by fatal diphtheria.....Snow, VIII-159
Larynx, traumatic atresia of, due to faulty intubation .....Northrup, XII-148

Discussion.—Miller, Cotton, Northrup, 150.

Laryngeal stenosis, persistent.....Griffith, XIII-272

Discussion.—Rotch, 275, 276; Buckingham, Koplik, Griffith, 276.

Laryngismus stridulus, death from, in insipient rickets.....Adams, IV-190

Discussion.—Huber, 195, 198; Earle, 195, 199; Blackader, 195; Caillé, 195, 196; Seibert, Koplik, 196; Jacobi, 196, 197, 199; Adams, 195, 196, 197, 199.

Lead poisoning, two fatal cases of.....Baines, XII-122
Lithemia, albuminuria a manifestation of.....Rachford, X-85

Lithemia, symptomatology of.....Rachford, IX-112
Discussion.—Holt, Jacobi, 128; Rotch, Crandall, Rachford, 129.

Liver, lesions of, in young children.....Freeman, XI-241
Discussion.—Wentworth, Koplik, Blackader, Freeman, 252.

Liver, marked enlargement of, in a case resembling typhoid fever.....Blackader, VIII-239
Loving cup—presentation to Dr. Rotch.....Yale, XIV-6
Lumbar puncture, for tuberculous meningitis.....Caillé, VIII-79
Lumbar puncture, experimental work on.....Wentworth, VIII-85
Lumbar puncture, a case of.....Jennings, VIII-109
Discussion.—Fruitnight, Blackader, 110; Holt, Caillé, Wentworth, 111.

Lung, abscess of.....Huber, III-235
Lung, abscess of, operation; recovery.....Huber, V-206
Discussion.—Huber, Rotch, Winters, Holt, 210.

Lungs, gangrene of, complicating typhoid fever.....Acker, VIII-141
Lungs, gangrene of.....Carr, XIII-256; Huber, XIII-263
Lungs, hemorrhage of, following exploratory puncture of the chest.....Koplik, XI-27
Lung, re-expansion of collapsed.....O'Dwyer, I-29
Discussion.—Northrup, 34, 38, 39; Jacobi, 38 39, 41, 42; Carr, 39; Jeffries, 42; O'Dwyer, 39, 42.

Lympho-sarcoma of the spleen, a case of.....Acker, VII-73
Lymphatism, enlarged bronchial nodes, a manifestation of.....West, XI-61
Lymph nodes, perforation of, into trachea; sudden death.....Caillé, XII-94
Malaria(?) an epidemic of atypical.....Freeman, XIII-224
Discussion.—Northrup, Packard, Kerley, 235; Freeman, 236, 237; Buckingham, Wentworth, Adams, 236.

Malaria, diagnosis and treatment of.....Vineberg, I-159
Discussion.—Holt, 182, 186; Fruitnight, 182, 183, 185; Jeffries, 182; Carr, 183, 186; Seibert, 183; Huber, 183, 185; Jacobi, 184, 187; Keating, 184; Vineberg, Caillé, 185; Watson, Latimer, 186.

Malaria, scarlet fever and meningitis, diagnosis of, from pneumonia.....Latimer, III-11
Malarial coma.....Acker, XII-163
Malarial, nephritis.....Kerley, X-140
Marasmus and infantile scurvy, a case of.....Huber, XIII-161
Masturbation (see Thigh-friction).

Maternal impressions, report of case.....Rachford, XIII-147

Discussion. — Rachford, Wentworth, 149.

Measles, a report on hospital cases.....Carr, X-194
Measles, a case complicated by appendicitis.....Williams, XIII-254
Measles, German, the enanthem of.....Forchheimer, X-118
Medication in pediatrics.....Earle, II-198

Discussion.—Carr, Holt, Seibert, Koplik, 203; Fruitnight, 204.

Melanosis, lenticularis progressiva.....Rotch, X-172
Meningitis, cerebro-spinal, in an infant.....Rotch, VII-67
Meningitis, cerebro-spinal.....Rotch, X-101
Meningitis, cerebro-spinal, followed by hydrocephalus .....Northrup, VIII-198

Discussion.—Osler, Caillé, 201, 202; Rotch, Wentworth, Christopher, Holt, Townsend, 201; Northrup, 202.

Meningitis, cerebro-spinal, a case of.....Rotch, X-101
Meningitis, tuberculous, early diagnostic symptoms of.....Northrup, II-69

Discussion.—Earle, 78; Rotch, 80, 86; Jacobi, 82; Winters, 84; Caillé, Fruitnight, 85; Smith, 86; Northrup, 78, 87.

Meningitis, tuberculous, with peculiar respiratory phenomena.....Chapin, X-219
Meningitis, tuberculous, (?) with recovery.....Acker, IX-75

Discussion.—Jacobi, Acker, 82, 83; Holt, 82; Brown, 83.

Meningitis, complicating pneumonia.....Holt, V-210

Discussion.—Smith, Rotch, Holt, 222.

Meningitis due to the typhoid bacillus.....Wentworth, XI-145

Discussion.—Koplik, Northrup, Freeman, Rotch, Wentworth, 153.

Meningitis, scarlet fever and malaria, diagnosis of, from pneumonia..... .....Latimer, III-11
Meningitis, lumbar puncture for tuberculous.....Caillé, VIII-79
Meningocele, spurious, traumatic cephalohydrocele, acquired cephalocele.....Huber, II-10

Discussion.—Fruitnight, 28.

Meningocele, a case of false.....Snow, V-174
Milk, breast, the clinical examination of.....Holt, IV-259
Milk, the filtration of, an aid to sterilization.....Seibert, VI-30; Kiliani, VI-34

Discussion.—Kiliani, 38; Crandall, Caillé, 39; Holt, 39, 40; Seibert, 39, 40, 41; Christopher, 40, 41.

Milk, preservation of.....Chapin, V-35
Discussion.—Seibert, 41; Rotch, Chapin, 41, 43; Koplik, 42; Caillé, 43.

Milk, low-temperature pasteurization of.....Freeman, VIII-113
Discussion.—Rotch, Holt, 123; Griffith, Freeman, 124.

Milk, heating of, for artificial feeding.....Freeman, X-35
Milk, the proteids of.....Chapin, XIV 149
Discussion.—Westcott, 152, 157; Chapin, 152, 153 159; Holt, 153; Buckingham, Miller, Cotton, 154; Koplik, Townsend, Edsall, 155; Freeman, 156; Wentworth, Griffith, 157.

Milk, the separation of bacteria from.....Freeman, XI-37
Discussion.—Cotton, Adams, Koplik, Forchheimer, Graham, Rotch, Freeman, 39.

Milk, breast, the management of, in cases of difficult digestion.....Rotch, II-88
Discussion.—Jacobi, Holt, 99; Fruitnight, Blackader, Rotch, 100.

Milk laboratories.....Rotch, IV-244
Discussion.—Koplik, 254. 258; Jackson, 255; Holt, 256; Jacobi, Rotch, 257, 258; Osler, 258.

Milk sugar in infant feeding.....Jacobi, XIII-150
Mirror writers.....Acker, VI-79
Monster *per defectum*.....Cotton, XIII-268
Mouth, nomenclature of diseases of.....Rotch and Forchheimer, VI-54
Myxedema, infantile.....Northrup, VI-109
Nasopharynx, treatment of, in scarlatina.....Seibert, XIII-64
Nasopharyngeal disease.....Huber, XII-81
Neapolitan children in America, the frequency of rickets among.....Snow, VI-160
Negroes, rickets in.....Acker, VI-137
Nephritis, acute, of malarial origin.....Kerley, X-140
Discussion.—Northrup, Caillé, Fruitnight, 144; Adams, Forchheimer, 145; Jacobi, Kerley, 146.

Nephritis, scarlatinal.....Smith, III-201
Nephritis, acute, primary.....Holt, III-223
Discussion.—Seibert, 230; 234; Forcheimer, Putnam, 231; Koplik, Smith, 231, 232; Carr, Fruitnight, Holt, Northrup, 232; Jacobi, 233, 234; Rotch, 234.
Nephritis, chronic parenchymatous, treated by renal decapsulation.....Caillé, XIV-120
Nephritis, chronic, in children and young adults.....Jackson, III-215
Nephritis, acute, following influenza.....Freeman, XII-132
Discussion.—Fruitnight, 137; Dorn-

ing, 137, 139; Jennings, 138; Rotch, 138, 139, 141; Carr, 139; Churchill, 140; Caillé, Freeman, 141.

Nephritis, acute, hemorrhagic, complicating influenza, with analysis of 40 cases.....Miller, XIII-193
Nerve centres, influence of venous congestion on spinal, reflex.....Rachford, VI-81
Neurosis, gastric.....Snow, V-185
Neuroses, the nutritional element in the etiology of.....Christopher, VI-149
Neuroses, physiological factors of.....Rachford, VII-18

Discussion. — Rotch, Holt, Jacobi, Rachford, 26.

Newly-born, acetanilid poisoning in, by absorption from umbilicus.....Snow, IX-39
Newly-born, inanition fever in.....Holt, VII-60
Newly-born, hemorrhagic disease of....Holt, XIII-278; Townsend, VI-47
Newly-born, respiratory failure, acute, recurring, in.....Snow, XIII-122
Newly-born, sarcoma of the cutis in.....Jacobi, IX-150
Newly-born, sclerema of.....Northrup, I-140
Newly-born, etiology of tetanus of.....Smith, VI-143
Newly-born, tetanus of.....Carr, VII-153; Smith, VII-192
Newly-born, tetanus of, a case resembling, without trismus.....Snow, VII-150
Nomenclature of diseases of the gastro-intestinal tract.....Rotch and Holt, VI-55
Nomenclature of diseases of the mouth...Rotch and Forchheimer, VI-54

Discussion.—Caillé 58, 59, 61; Holt, 59, 60, 61; Rotch, 59; Seibert, 59, 60; Christopher, 61.

Nutritional element in the etiology of neuroses .....Christopher, VI-149
Nystagmus and choreic movements of the head associated with rickets.....Caillé, I-237

Discussion.—Rotch, 239.

O'Dwyer's tubes, with a report of 350 cases of intubation.....Brown, II-182
Osteitis, tuberculous, of the hip .....Keating, III-69
Osteomyelitis, a case of multiple, in an infant.....Rotch, XI-208
Otitis, followed by fatal cerebral abscess.....Fruitnight, XII-56
Otitis media, purulent.....Lockwood. VII-129

Discussion.—Caillé, Lockwood, 135;

Ozone, the value of nascent, with demonstration of generator.....Caillé, IV-14

Discussion.—Holt, 19; Rotch, 20; Northrup, 20, 21; Blackader, Fischer, 21; Rachford, 22; Caillé, 19, 20, 21, 22.

Pancreatic digestion of casein.....Rachford, XII-29

Discussion.—Rotch, 42, 43, 44; Rachford, 42, 44; Holt, 43, 45; Fruitnight,

Blackader, 43; Caillé, 43, 44; Miller, Koplik, 44.

Papillomata, multiple, of the larynx; tracheotomy; improvement; death from diphtheria following.....Snow, VIII-159

Discussion.—Northrup, Rotch, Holt, Caillé, Seibert, 162.

Paralysis, epidemic.....Chapin, XII-158

Discussion.—Adams, Griffith, Chapin, 162.

Parapleiga, spastic, two cases of.....Latimer, I-195
Pediatrics, development the keynote of.....Christopher, XIV-9
Pediatrics, its relation to general medicine.....Jacobi, I-6
Pediatric poem, A.....Forchheimer, VII-7
Pediatrics, iconoclasm and original thought in the study of.....Rotch, III-6
Pediatric literature, the evolution of, in the United States.....Adams, IX-5
Pediatrics, methods of instruction in.....Northrup, XI-7

Discussion.—Rotch, 15, 17, 21, 24; Crandall, 17, 25; Griffith, 17, 26; Chapin, 18, 20; Blackader, 19; Forchheimer, 20, 21; Adams, 21; Cotton, 23. 24, 25.

Pericardium, adherent, four cases of.....Swift and Freeman, VIII-171
Pericardium, adherent, with marked hypertrophy of the heart, chronic peritonitis and recurring ascites.....Osler, VII-214
Pericarditis, adhesive.....Holt, VIII-169
Peritonitis, chronic, with marked hypertrophy of the heart, recurring ascites and adherent pericardium.....Osler, VII-214
Peritonitis, general, following appendicitis.....Holt, VIII-229

Discussion.—Osler, 231.

Peritonitis, following vulvo-vaginal catarrh.....Huber, I-124

Discussion.—Caillé, Koplik, 127.

Peritonitis, suppurative.....Reid, II-204
Peritonitis, tuberculous, followed by umbilical fistulæ.....Acker, XIV-110
Peritonitis, tuberculous, operative treatment of.....Caillé, XII-17

Discussion.—Fruitnight, 24; Rotch, 25, 27; Cotton, 26; Jackson, Koplik, Caillé, 27.

Peritonitis, tuberculous, treatment of.....Rotch, XIV-102

Discussion.—Adams, Acker, Forchheimer, 105; Chapin, Caillé, 106; Koplik, 106, 108; Cotton, 107; Jacobi, 107, 109; Miller, Seibert, 108; Rotch, 105, 108, 109.

Perityphlitis in the young.....Fruitnight, III-85

Discussion.—Putnam, 98.

Peroxid of hydrogen, notes on.....Jacobi, IV-208

Discussion.—Caillé, 212, 213; Blackader, 213; Seibert, Buckingham, 214; Koplik, Huber, Earle, 215; Jacobi, 216.

Peroxid of hydrogen, its value in diphtheria.....Smith, V-17
Pertussis, complications of.....Chapin, V-126
Pertussis, cough in influenza, simulating.....Forchheimer, XII-152
Pertussis, heart strain in.....Koplik, V-90
Pertussis, local treatment of.....Griffith, V-110
Pertussis, constitutional treatment of.....Forchheimer, V-116

Discussion.—Osler, 120; Holt, 121; Northrup, Fruitnight, 122; Winters, 123; Seibert, 124; Caillé, Smith, 125.

Pharyngo-esophageal stenosis, congenital.....Adams, VIII-166
Pilocarpin in the treatment of scarlet fever.....Saunders, XIV-181
Playrooms, sun, on city roofs.....Northrup, XIII-220

Discussion.—Putnam, 223; Northrup, Adams, Winters, 223, 224; Rotch, Griffith, 224.

Pneumonia, the diagnosis of, in infancy and early childhood:
1.—Diagnostic symptoms and signs of lobar pneumonia and diagnosis from meningitis, malaria, scarlet fever, etc.....Latimer, III-11
2.—Diagnosis of consolidation of the lung from effusion.....Forchheimer, III-18
3.—Diagnosis of tuberculous broncho-pneumonia .....Osler, III-25
4.—Temperature in pneumonia.....Holt, III-30
5.—Diagnosis of broncho-pneumonia from bronchitis .....Holt, III-48

Discussion.—Koplik, 51; Jacobi, 52, 53, 55, 56, 57; Smith, Forchheimer, 55, 57; Holt, 55, 56; Rotch, Northrup, 56; Adams, 57.

Pneumonia, a case probably due to streptococcus infection.....Carr, XIV-205

Discussion.—Adams, Carr, 209.

Pneumonia, temperature curve in acute, croupous .....Jennings, XIV-201

Discussion—Morrill, 204.

Pneumonia, croupousa, scarlatina anginosa, amnesic aphasia.....Adams, VII-136
Pneumonia, frank (lobar), an analysis of 100 cases of.....Morrill, IX-144
Pneumonia, with meningitis complicating.....Holt, V-210

Pneumonia, two cases of prolonged.....Crandall, X-186
Poisoning, acetanilid, by absorption.....Snow, IX-39
Poisoning, arsenical.....Morse, XIII-119
Poisoning, carbolic acid.....Adams, VI-131
Poisoning, lead, two fatal cases of.....Baines, XII-126
Poisoning, vapo-cresolene.....Adams, XII-174
Polypi, rectal, etiology of.....Huber, XIII-112
Porencephalus .....Acker, XI-131
Pott's disease, diagnosis of.....Brown, VI-89

Discussion.—Rotch, 104; Holt, 105.

Prenatal infection in diseases of infancy.....Davis, IX-92
Prolapsus-recti due to large stone in the bladder .....Caillé, I-157

Discussion.—Fruitnight, 158.

Proctitis in early infancy.....Starr, V-58

Discussion.—Fruitnight, 62; Christopher, 63; Starr, Jacobi, 63, 64; Koplik, 64.

Progressive muscular atrophy with Jacksonian epilepsy and endothelioma of the cerebral membranes.....Packard, VIII-147
Puncture of the chest, exploratory, pulmonary hemorrrhage following.....Koplik, XI-27
Purpura, a fatal case, with notes on literature .....Jackson, II-158

Discussion.—Earle, 164, 166; Booker, 164; Jacobi, 165, 166; Koplik, Smith, Fruitnight, 167; Rotch, 166.

Pyelitis, acute, three cases of.....Holt, VI-117

Discussion.—Osler, 132; Seibert, Caillé, Holt, 133.

Pygophagus, Dr. S. Tynberg's, with histories of 13 other cases.....Jacobi, VII-121
Pyloric stenosis in infants, with report of cases .....Saunders, XIII-282

Discussion.—Holt, 293.

Pyopneumothorax, a case of.....Carr, VII-69
Pyothorax, the significance of fever following operation for.....Caillé, X-54
Rectal polypi, etiology of.....Huber, XIII-112
Reflex centres, influence of venous congestion on spinal.....Rachford, VI-81
Renal complications of acute enteric diseases.....Morse, XI-40

Discussion.—Koplik, Adams, 58; Chapin, Cotton, Northrup, Morse, 59.

Respiration, artificial, forcible.....O'Dwyer, III-128

Discussion.—Northrup, Booker, O'Dwyer, 132.

Respiration, noisy, in children.....Brown, I-43
Discussion.—Fruitnight, Caillé, Huber, Carr, 55.

Respiratory failure, acute, recurring, in the newly-born .....Snow, XIII-122
Respiratory phenomena, peculiar, in a case of tuberculous meningitis.....Chapin, X-219
Rest, necessity of prolonged, after cardiac inflammations .....Holt, XI-183
Retro-esophageal abscess, a case of.....Griffith, IX-169
Discussion.—Chapin, Koplik, 177; Rotch, 178; Adams, Griffith, 179.

Rhachischisis, a case of.....Rotch, XII-47
Rheumatic children, the mangement of.....Crandall, XIV-59
Discussion.—Chapin, Jacobi, 68; Carr, Koplik, Wilson, 69; Packard, 69, 71; Blackader, 70; Crandall, 71.

Rheumatism, acute, articular, in infants under one year..... .....Miller, XI-79
Discussion.—Crandall, 90, 91; Jennings, Miller, 90; Koplik, Griffith, Cotton, 91.

Rheumatism and chorea, the relation of, Townsend, IV-217; Crandall, IV-225; Adams IV-231
Discussion.—M. Allen Starr, 238; Jacobi, 239; Holt, Townsend, 240, 241; Booker, Fischer, 241; Osler, 242; Crandall, 243.

Rickets, associated with nystagmus and choreic movements of the head.....Caillé, I-237
Rickets, congenital, a case of.....Townsend, VI-106
Rickets, etiology of.....Smith, V-195
Rickets, head-nodding and head-rotation in.....Miller, XII-72
Rickets, incipient, death from laryngismus stridulus in.....Adams, IV-190
Rickets, its frequency among Neapolitan children in America.....Snow, VI-160
Discussion.—Griffith, 171, 172; Holt, 171, 174; Fruitnight, 172, 174; Seibert, 172, 174, 175; Adams, Crandall, 173; Koplik, 173, 174, 175; Stowell, Northrup, 174; Carr, 175; Acker, Snow, 176.

Rickets, its treatment with lacto-phosphate of lime.....Fruitnight, V-168
Discussion.—Seibert, Fruitnight, Smith, Rotch, Winters, 174.

Rickets in negroes.....Acker, VI-137
Rickets, some manifestations of, without severe bone lesions.....Carr, II-33

Rotch, Thomas M., loving cup presentation to.....Yale, XIV-6
Sacro-coccygeal tumor, operation; recovery.....Huber, IV-200; X-209
Sarcoma of the cerebellum.....Adams, XI-69
Sarcoma of the cutis of the newly-born.....Jacobi, IX-150
Discussion.—Carr, 156; Freeman, Northrup, Jacobi 157.
Sarcoma of the kidney.....Warner, II-65; Churchill, X-178
Sarcoma of the kidney treated with erysipelas toxin.....Fruitnight, VII-168
Sarcoma of the skin of the back.....Carr, V-204
Sarcoma of the suprarenal capsule.....Caillé, VII-75
Discussion.—Holt, Caillé, 76.
Sarcoma of the thymus and bronchial glands.....Acker, VIII-214
Scarlet fever, general prophylaxis in.....Smith, II-129
Scarlet fever, prophylaxis as to complications and sequelæ.....Smith, III-58
Discussion.—Fruitnight, 69.
Scarlet fever, malaria and meningitis, diagnosis of, from pneumonia.....Latimer, III-11
Scarlet fever followed by organic heart disease.....Acker, VIII-163
Scarlet fever followed by symmetrical gangrene.....Wilson, VII-109
Scarlet fever and its complications, treatment of.....Fruitnight, I-95
Scarlet fever, pilocarpin in the treatment of.....Saunders, XIV-181
Scarlatina, anomalous.....Starr, VII-90
Scarlatina, eruptions simulating.....Blackader VII-76
Discussion.—Griffith, 104; Caillé, Chapin, 106; Dorning, Jacobi, Wilson, 107; Adams, Fruitnight, Macdonald, Acker, 108; Starr, 109.
Scarlatina anginosa, pneumonia crouposa and amnesic aphasia.....Adams, VII-136
Scarlatina, treatment of naso-pharynx in.....Seibert, XIII-64
Scarlatinal nephritis.....Smith, III-201
Scarlatiniform, erythema.....Jennings, VII-84
Scarlatinal, pseudo-diphtheritic angina, its relation to diphtheria.....Booker, IV-45
Sclerema neonatorum.....Northrup, I-140
Discussion.—Huber, Koplik, Holt, Jacobi, 146; Northrup, 147.
Scleroderma, acute, diffuse.....Barker, V-142
Discussion.—Northrup, Jacobi, 159; Koplik, 160.
Scorbutus in infants, American cases.....Northrup, III-99
Discussion.—Holt, Fruitnight, 116; Rotch, 116, 119; Northrup, 116, 118, 119; Jacobi, 117, 118, 119, 120; Smith, 120.

Scorbutic membranous colitis.....Fruitnight, XI-212
Scurvy, infantile, diagnosis of.....Fruitnight, VI-10
Discussion.—Seibert, 29; Crandall, 30.
The Pediatric Society's Collective Investigation:
Majority report.....Griffith, Jennings, Morse, X-5
Minority report.....Caillé, X-24
Discussion.—Christopher, 24, 25; Griffith, 24, 31; Northrup, 24; Morse, 25, 28; Jennings, 25, 30; Booker, 25, 26, 31; Caillé, 25, 30; Forchheimer, 25; Buckingham, 26; Winters, 28; Jacobi, Adams, 29; Holt, 31; Freeman, 32.
Scurvy, infantile.....Huber, XI-215
Scurvy, in an infant of six weeks.....Crandall, XI-173
Discussion. — Dorning, 179; Rotch, Griffith, Koplik, Wentworth, Adams, Northrup, Crandall, 181.
Scurvy, a case of, with marasmus.....Huber, XIII-161
Skin (see Cutis).
Skin of the back, sarcoma of.....Carr, V-204
Specialism .....Osler, IV-6
Spine, metastatic sarcoma of, with sarcoma of the suprarenal capsule.....Caillé, VII-75
Spleen, extreme enlargement of, with anemia, a case of.....Hamill, XIV-76
Discussion.—Wentworth, Martin, Christopher, 89; Hamill, 90.
Spleen, lympho-sarcoma of.....Acker, VII-73
Stethoscope, a new binaural.....Koplik, XI-171
Stomach, the capacity of, in infancy.....Holt, II-168
Discussion.—Rotch, 175, 176, 177; Seibert, 175; Earle, 176; Jacobi, 177; Holt, 176, 177.
Stomatitis aphthosa, the etiology of.....Forchheimer, III-243
Discussion.—Koplik, 252; Putnam, 253, 254; Rotch, 253, 255; Adams, 253; Forchheimer, 254.
Streptococcus infection, a probable cause of pneumonia...Carr, XIV-205
Sugar of milk in infant feeding.....Jacobi, XIII-150
Summer diarrhea (see Diarrhea).
Summer diarrhea, bacteria of.....Booker, I-198
Summer diarrhea, a chemical study of.....Vaughan, II-109
Summer diarrhea, the early history of.....Booker, XIII-7
Summer diarrhea, a study of.....Jeffries, I-249
Summer diarrhea, a study of 555 cases of.....Kerley, XIII-36
Summer diarrhea, treatment of.....Townsend, II-101
Submersion, irrigation by, in empyema.....Adams, X-80
Suprarenal capsule, hemorrhage into.....Hamill, XII-176
Suprarenal capsule, sarcoma of, with metastatic sarcoma of the spine.....Caillé, VIII-75

Syphilitic broncho-stenosis.....Seibert, IV-180
Temperature curve in acute, croupous pneumonia.....Jennings, XIV-201
Temperature in pneumonia.....Holt, III-30
Temperature in pulmonary tuberculosis.....Adams, XIII-184
Temperature, prolonged, high, with double sacculated empyema.....Holt, XIII-214
Testis, tuberculosis of.....Koplik, I-128
Teratoma of the sacrum and coccyx.....Huber, X-209

Discussion.—Jacobi, Huber, 217.

Tetanus neonatorum, etiology of.....Smith, VI-143
Tetanus neonatorum, a case of.....Carr, VII-153
Tetanus neonatorum, symptoms and treatment of.....Smith, VII-192
Tetanus neonatorum, a case resembling but without trismus.....Snow VII-150
Tetanus, Escherich's pseudo, in diphtheria.....Snow, XIV-210
Tetany, carpo-pedal contractions, a manifestation of.....Earle, I 153
Tetany, two cases of.....Crandall, VII-208
Thigh-friction in infants.....Townsend, VIII-186

Discussion.—Adams, 188; Yale, Wentworth, Holt, Booker, Griffith, Townsend, 189.

Throat and ears, the care of.....Northrup, V-129
Thymus gland, sudden death due to compression of large blood vessels by an enlarged.....Caillé, XIV-194

Discussion.—Jacobi, 194, 195; Caillé, 194; Futnam, 195.

Thymus and bronchial glands, sarcoma of.....Acker, VIII-214
Thyroids, their use in diseases other than cretinism.....Koplik, IX-65
Tic convulsif, a case of.....Wilson, IX-165
Tics or habit movements, classification of.....Osler, VIII-215
Tonsillar infection, remote diseases arising from.....Forchheimer, XIV-91

Discussion.—Jacobi, Freeman, 99; Christopher, 100; Jennings, Forchheimer, 101.

Tonsillotomy followed by diphtheria and croup.....Caillé, VI-66

Discussion.—Jacobi, 71, 73, 74; Koplik, 71; Fruitnight, 73; Brown, 72; Caillé, Rotch, 74.

Trachea, perforation into by tuberculous bronchial lymph node.....Caillé, XII-94

Discussion.—Rotch, Caillé, 94, 95; Freeman, 95; Dorning, 95, 97; Miller, Blackader, Fruitnight, 96; West, 96, 97.

Tremor, unilateral, two cases of.....Griffith, IX-158

Discussion.—Rotch, 162, 164; Holt, 163; Griffith 164.

Tuberculosis, the anemia of.....Rachford, IV-151

Discussion.—Fischer, 173; Osler, Rotch, Rachford, 174.

Tuberculous (?) meningitis with recovery.....Acker, IX-75
Tuberculous peritonitis, treatment of.....Rotch, XIV-102
Tuberculous peritonitis, the operative treatment of.....Caillé, XII-17
Tuberculosis in children.....Osler, V-178
Tuberculosis, its direct transmission to fetus.....Keating, V-47

Discussion.—Jacobi, 58.

Tuberculosis, temperature in.....Adams, XIII-184

Discussion.—Griffith, Adams, 191, 192; Wentworth, 191; Holt, 192.

Tuberculosis, treatment of.....Rachford, XIII-166
Tuberculosis of the testis.....Koplik, I-128
Tumor, multiple of the brain.....Chapin, VIII-236
Tumor, sacro-coccygeal.....Huber, IV-200
Typhoid bacillus, a cause of meningitis.....Wentworth, XI-145
Typhoid fever (see Enteric Fever).
Typhoid fever, its occurrence under two years.....Northrup, IV-109; VII-205
Typhoid fever, in infancy and childhood.....Earle, IV-112; Blackader, XII-98
Typhoid fever, in infancy.....Christopher, IV-134

Discussion.—Koplik, 137; Jacobi, 137, 138, 146, 147, 148; Northrup, 137, 138, 139, 143, 146; Blackader, 138; Winters, 139; Huber, 141, 143; Seibert, 141; Adams, 143; Rotch, 147; Earle, 149.

Typhoid fever, with gangrene of the lungs.....Acker, VIII-141
Typhoid fever, a case resembling, with marked enlargement of the liver.....Blackader, VIII-239

Discussion.—La Fleur, Seibert, 243.

Typhoid fever, exclusive soup diet and rectal irrigations in.....Seibert, XII-118
Typhoid fever followed by temporary insanity.....Adams, VIII-177
Typhoidal appendicitis.....Seibert, XIV-115

Discussion.—Caillé, Seibert, 119.

Umbilical fistulæ due to tuberculous peritonitis.....Acker, XIV-110
Umbilicus, acetanilid poisoning by absorption from.....Snow, IX-39
Urine, autointoxication with neutral magnesium phosphate in.....Jennings, XIV-184

Discussion.—Edsall, Christopher, 189.

Urine, incontinence of.....Rachford, V-161; Huber, XI-163

Discussion.—Rotch, Crandall, Adams, V-167; Griffith, 168.

Urine of healthy children.....Churchill, X-91

Discussion.—Christopher, Tyson, Fruitnight, Rotch, Griffith, 99; Churchill, 99, 100; Holt, 100.

Urticaria of the mucous membranes, especially associated with asthma.....Packard, XI-92

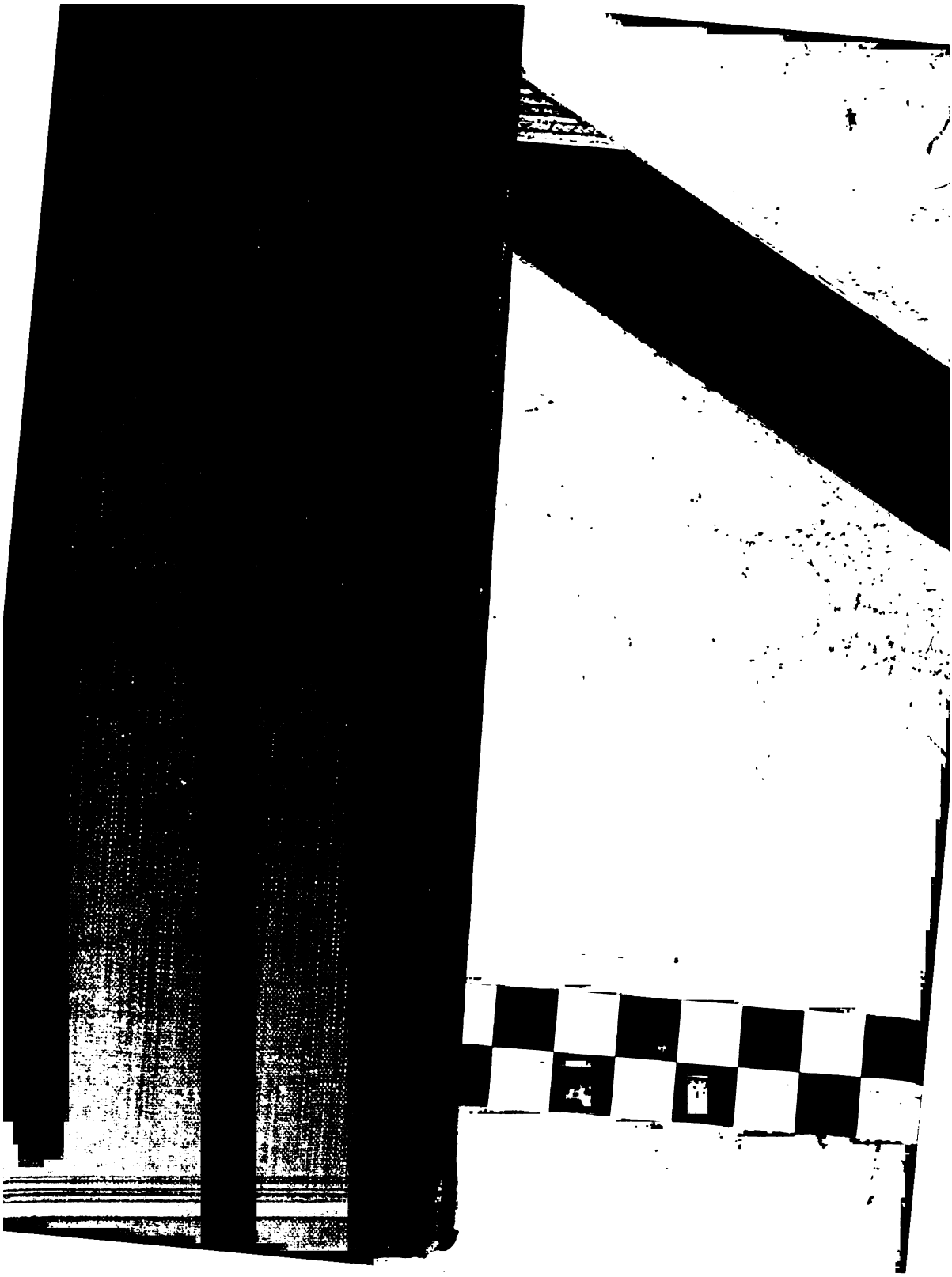
Discussion. — Blackader, Wentworth, Miller, Dorning, Packard, 109.

Vaccinoid.....Forchheimer, XI-155

Discussion.—Putnam, Freeman, 161; Dorning, Forchheimer, 162.

Vapo-cresolene, poisoning by.....Adams, XII-174
Varicella gangrenosa.....Lockwood, IX-131
Venous congestion, influence of, on the spinal reflex centres.....Rachford VI-81

Discussion.—Rotch, Fruitnight, 88.

Viscera, transposition of, with malformation of the heart.....Griffith, XI-32
Vomiting, gavage in the treatment of persistent.....Kerley, III-137
Vulvo-vaginal catarrh followed by peritonitis.....Huber, I-124
Vulvo-vaginitis, gonorrheal, with multiple arthritis.....Acker, XIII-238
Weight, increase of, in infants artificially fed.....Koplik, XI-110
Wry-neck associated with marked facial asymmetry.....Osler, III-133

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